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Table of Contents.

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	Page.
ORIGINAL ARTICLES—	
An Investigation into the Incidence of Pulmonary Tuberculosis in Young Women of Adelaide in the Age Group Fifteen to Thirty Years, by H. W. Wunderly	229
A Study of the Tuberculous Cavity of the Lung, by Roy Le P. Muecke, M.B., B.S.	232
Tuberculosis as a Problem for the State, by Cotter Harvey	239
REPORTS OF CASES—	
Melanotic Sarcoma in a Native of New Britain, by A. V. G. Price	241
REVIEWS—	
The Sheep's Brain	242
NOTES ON BOOKS, ETC.	242
LEADING ARTICLES—	
The Tuberculosis Problem in Australia	243
CURRENT COMMENT—	
The German Measles and the Recently Prevalent Sore Throat	245
Prothrombin Content of Preserved Blood	245
ABSTRACTS FROM MEDICAL LITERATURE—	
Physiology	246
Biological Chemistry	247
SPECIAL ARTICLES ON PSYCHIATRY IN GENERAL PRACTICE—	
Confusional Insanity	248
NAVAL, MILITARY AND AIR FORCE—	
A Clinical Meeting in Palestine	249
Appointments	249
CORRESPONDENCE—	
Ménière's Syndrome and Gout	250
Reorganization of the Medical Profession	251
AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED—	
Annual Meeting	251
AUSTRALIAN MEDICAL BOARD PROCEEDINGS—	
South Australia	251
New South Wales	252
NOMINATIONS AND ELECTIONS	252
MEDICAL APPOINTMENTS	252
BOOKS RECEIVED	252
DIARY FOR THE MONTH	252
MEDICAL APPOINTMENTS: IMPORTANT NOTICE	252
EDITORIAL NOTICES	252

AN INVESTIGATION INTO THE INCIDENCE OF PULMONARY TUBERCULOSIS IN YOUNG WOMEN OF ADELAIDE IN THE AGE GROUP FIFTEEN TO THIRTY YEARS.[1]

By H. W. WUNDERLY,

Adelaide.

The Mantoux Test.

THE technique used for the Mantoux test was that recommended by the committee of the National Health and Medical Research Council. The tuberculin used was old tuberculin supplied by the Commonwealth Serum Laboratories; 0·1 cubic centimetre of a 1 in 1,000 solution was injected into the flexor aspect of the forearm and the reaction was read in forty-eight hours. During the first three months this dilution was made by the Commonwealth Serum Laboratories; but after October, 1938, the dilution was prepared every week from old tuberculin supplied by the Commonwealth Serum Laboratories.

In most instances only one test was performed; but during the course of the survey these reactions were compared with reactions obtained with the following: (*a*) a 1 in 1,000 solution of old tuberculin supplied by Bayer Pharma, (*b*) dilutions of old tuberculin made up to three months previously by the Commonwealth Serum Laboratories, (*c*) purified protein derivative supplied by the Commonwealth Serum Laboratories, and purified protein derivative supplied by the firm of Sharpe and Dohme.

The result of this small investigation was to confirm the results obtained by Dr. Cotter Harvey and Dr. Douglas Anderson in Sydney. In no instance was a positive reaction obtained with the German tuberculin when no reaction had been obtained with the tuberculin of the Commonwealth Serum Laboratories. In the small series of subjects investigated, the three months' old dilution remained active and a reaction was obtained with a seven months' old dilution. The reactions obtained with the first dose of purified protein derivative were neither so sharp nor so easy to interpret as those obtained with the tuberculin supplied by the Commonwealth Serum Laboratories.

[1] Aided by a grant from the National Health and Medical Research Council.

Classification of Reactions.

At the end of three months the classification of reactions was altered and the classification suggested by the workers at the Henry Phipps Institute was adopted. This classification is as follows: a "+" reaction consists of an area of swelling five to ten millimetres in diameter; a "++" reaction consists of an area of swelling 10 to 20 millimetres in diameter; a "+++" reaction consists of an area of swelling over 20 millimetres in diameter; a "++++" reaction consists of an area of swelling exceeding 20 millimetres in diameter, with vesiculation or central necrosis.

There was no obvious correlation between the strength of the tuberculin reaction and the condition of the lungs as revealed by the skiagram. It appears that the severe reactions occur in those who are constantly being exposed to infection.

One interesting case was that of a nurse who had been looking after a patient with an open pulmonary tuberculous lesion. From being a non-reactor to the Mantoux test she became a reactor, *erythema nodosum* developed, and she was the only instance of a "++++" reactor showing radiological evidence of pulmonary infiltration.

Development of Allergy.

It is generally recognized that in primary pulmonary tuberculous infection there is an interval of three to seven weeks before the response to the Mantoux test becomes "positive". It appears that differences in the number of infecting bacilli and in the clinical character of the disease do not influence the incubation period; nor can it be held that the duration of the incubation period is directly proportional to the severity of the disease; in other words, a long incubation period does not mean that the disease will be benign, nor does a short incubation period always accompany a malignant infection.

The Value of the Mantoux Test.

The survey undertaken was a case-finding survey, and the Mantoux test was used as a screen to find the reactors. The error is so small (2% reported by D'Arcy Hart in 1932) that it was considered unnecessary to take X-ray photographs of those who failed to give a reaction. If, in spite of an absence of reaction, there was a history of close contact with an infective patient, or if the subject complained of cough, expectoration, unexplained loss of weight or increasing lassitude, she was examined by means of fluoroscopy and sometimes an X-ray photograph was taken. In this series none of those who failed to react to the Mantoux test gave radiographic evidence of pulmonary tuberculosis. Many who had a suspicious history were retested with a 1 in 100 solution when they failed to react to the weaker solution.

The limitations of the Mantoux test are that a negative response would occur during the interval of three to seven weeks while allergy was developing. It is also stated that those who have completely healed calcified areas in the lymphatic glands do not react to the Mantoux test.

There may be no reaction to tuberculin, in spite of tuberculous infection, in the following circumstances: (i) in miliary tuberculosis; (ii) in extensive advanced phthisis; (iii) in tuberculous peritonitis and, according to Long (1939), in pleurisy with effusion; (iv) in measles and scarlet fever; (v) after a foregoing reaction to tuberculin; (vi) immediately after irradiation of the skin with a full erythema dose. When these limitations have been admitted it is still possible to define two groups of subjects: (*a*) those who have never been infected and give no reaction, (*b*) those who are infected and give a reaction.

The Results of the Mantoux Test.

Of the 1,273 reactors to the Mantoux test, 868 were classified as having no radiological evidence of pulmonary tuberculosis, past or present. In view of the findings in similar surveys it is my intention to reexamine all the films. In many cases it was difficult to decide whether some shadows in the peripheral half of the fields were due to small areas of calcification or whether they were shadows of blood vessels seen "end on". Further, in collecting the figures for the report of October 2, 1939, I do not include the presence of "pleural caps" as evidence of past or present tuberculosis.

There are several reasons why there may be no radiological evidence of past or present infection of the lungs or regional lymph glands in subjects who react to the Mantoux test. First of all, the primary infection could have been extrathoracic—for example, in the bowel or tonsil. Changes occur in the primary lesion in the lung, and while many of these lesions remain as calcified areas, others are converted into bone with marrow tissue and some are completely absorbed. It is probable that in many cases the primary lesion is situated in an area which is not visible on an ordinary postero-anterior film, being hidden by the shadow of the heart or great vessels.

During the course of this survey five nurses who had towards the end of 1938 failed to react to the Mantoux test were exposed to infection in the course of their nursing duties. When retested some months later, all yielded a skin reaction; one suffered from *erythema nodosum* and had radiological evidence of pulmonary infiltration, one had *erythema nodosum* and normal-looking lungs, one had bilateral pulmonary tuberculosis with the mycobacterium of tuberculosis in her sputum; the remaining two gave no radiological evidence of pulmonary involvement.

Seventy-three nurses belonging to the age group fifteen to thirty years were investigated at the mental hospitals; 54 gave a reaction to the test, while the remaining 19 failed to react. One nurse had an active open lesion with sputum containing tubercle bacilli, and several had healed lesions. In the mental hospitals those of the patients who are suffering from pulmonary tuberculosis are isolated; but it is possible that there are many unrecognized sources of infection. Moreover, it is almost impossible to make most patients in mental hospitals take even elementary precautions. As a result of this piece of investigation and of the experience of men like Heimbock, I suggested to the Director-General of Medical Services that it would be wise to keep nurses who failed to react to the Mantoux test away from patients with pulmonary tuberculosis, and that all patients entering mental hospitals should have their lungs radiologically examined on their admission. This would help with the diagnosis of many infectious cases which might otherwise escape detection. It will be noticed that of these nurses working in mental hospitals the percentage who gave a skin reaction was three times as great as that of the nurses in other hospitals.

Radiological Examination of the Lungs.

As "case-finding" was the real object of this survey, it was important to detect all those subjects whose X-ray photographs contained shadows which could be produced by active or by latent pulmonary tuberculosis. The chief attention was centred on this aspect of the investigation; but at a later date a reexamination will be made of the films and an attempt will be made to classify all the lesions.

Only in exceptional cases was it held that a lesion could be classed as "active" on the appearances in one X-ray photograph. The presence of cavitation was accepted as a sign of activity. In other cases the examination of a series of photographs, the clinical history and clinical examination and the results of laboratory investigation were all taken into account.

During the early weeks it was thought that all "++++" reactors should be radiologically examined at frequent intervals; but it soon became evident that these were not the ones in whose X-ray photographs abnormalities were apparent. Assmann makes the following statement:

> Only in extremely rare instances of cases verified by positive sputum tests does the radiological examination reveal a complete absence of visible change, and in these the density and extent of the lesions may have been too slight to allow of their recognition, or other shadows may have hidden them. The reverse is, however, a matter of daily experience, that tuberculous disease can be demonstrated radiologically when all other clinical means fail.

It was necessary to adopt a classification of the lesions, and the following was used: (*a*) healed and probably healed, (*b*) probably active. Those subjects who had no definite evidence of previous infection with tuberculosis were described as "negative". Earlier in this report I mentioned the difficulty of classifying some of the small round shadows in the lateral half of the lung fields. Some were undoubtedly due to spots of calcification; but others, especially when a vessel shadow could be seen continuing on from the round spot, were regarded as possibly due to a blood vessel "end on". The presence of thickened pleura over the apex, a "pleural cap", often accompanied by puckering, was not accepted as definite evidence of previous tuberculous infection.

A better classification is that given by Kayne, Pagel and O'Shaughnessy in their book on pulmonary tuberculosis:

> A pulmonary tuberculous lesion when first recognized must now be classified into one of four types: healed, active progressive, active regressive, or latent. A healed lesion should imply a lesion showing no morbid tissue activity and containing no tubercle bacilli. Nevertheless, many calcified lesions do contain bacilli, and recrudescence of disease in their vicinity is not a rare phenomenon. In regard to the term "active", it should be noted that pathological tissue activity may be either progressive or regressive in regard to the disease process. From a clinical point of view it is important to recognize to which of these phases a newly discovered tuberculous lesion belongs. Finally, a latent lesion may be defined as a non-progressive lesion, giving rise to no symptoms but containing within it those conditions of bacillus and host that are favourable to recrudescence of the focus, e.g. an encapsulated caseous focus containing tubercle bacilli.

It is admitted that in this survey, included in the group "healed and probably healed", are some conditions which should have been classified as "latent". In some instances a second X-ray photograph taken after an interval of two or three months revealed an alteration in the appearance of the lesion or lesions, and this alteration, whether towards progression or regression, was considered evidence of activity. In other instances it was necessary to have

a series of four or five X-ray photographs before it could be decided whether a lesion was really healed or merely "latent". As the investigation progressed it became increasingly evident that the apical lesion was dangerous. This is the opinion of Saye and of several Scandinavian workers. Another point which emerged was that an early active tuberculous lesion can occur in persons with a low blood sedimentation rate.

Of the 1,273 subjects who reacted to the Mantoux test, 61 were finally reported as having active pulmonary tuberculosis. These reports were sent to medical practitioners in private practice and they undertook the necessary treatment. As was pointed out by D'Arcy Hart in his Milroy Lecture of 1937: "The object of a survey is to help prevent the development of disabling pulmonary tuberculosis and to help prevent the spread of infection to others." D'Arcy Hart went on to state that "on the average, a slight and early case does better with, than without, treatment, and with treatment it does better than a later and more extensive case".

These 61 young women were all at full work and were supposed to be in good health. In all cases the diagnosis was made when their disease was in a curable stage and all were saved from contracting disabling pulmonary tuberculosis. In addition, in the search for the sources of infection, many persons suffering from pulmonary tuberculosis in an infectious stage were detected amongst the members of the community. In some instances it was found that infection had occurred at the place of employment, but in the majority it had occurred in the home.

Two important questions remain to be answered: (i) Why is the incidence of pulmonary tuberculosis so high among young women of the age group fifteen to thirty years? (ii) Why is the incidence so high in Adelaide compared with other cities of the Commonwealth?

I believe the answer to the first question is contained in the report of surveys conducted in Detroit and New York City and published by the National Tuberculosis Association:

> The findings of the two studies seem to overthrow most of the existing theories on the subject, viz., the increasing industrialisation of women, the desire for a slender figure, flimsy dress, cigarette smoking, and the excesses of the so-called "jazz age". The proponents of these theories lost sight of the fact that the problem had existed long before the manifestations of any of these causes. There is no reasonable basis for believing that the increased tuberculosis mortality among young women is due to insufficient clothing, dieting, poor food habits, consistent lack of sleep, or unusual recreational habits.
>
> We believe, although it cannot be statistically proved, that the psychic and the physical changes of adolescence and early adult life cause young women to be unusually susceptible to tuberculosis and constitute the fundamental reasons for their high mortality rates.

It is not possible to be any more definite when an attempt is made to answer the second question. Many of these young women came from quite comfortable homes. The conditions of overcrowding, lack of proper nourishment and poor housing conditions are not so evident in Adelaide and its suburbs as they are in some industrial areas in the Commonwealth. It does appear that the population must be exposed to contact with a large number of "open cases" of pulmonary tuberculosis. The evidence of "infection", as shown by a reaction to the Mantoux test, was highest amongst those working on the ground floor of the large stores. There was also a high rate amongst the lift attendants. Ever since the war of 1914-1918 the patients from the sanatoria nearest the city have been allowed week-end leave. But it is not only during the week-ends that patients with "open" lesions are free to mix with the general public, attend picture theatres, take their meals at any restaurant and frequent all public buildings, including public houses. In addition, the standard of "arrest" of the disease has not been sufficiently high; officially, an absence of tubercle bacilli from the sputum at two examinations has been accepted as sufficient evidence of non-infectivity. I am glad to report that efforts are being made to raise this standard; cultural methods for sputum examination are being introduced, and the tuberculosis service at the chest clinic of the Royal Adelaide Hospital gives both practitioner and patient not only "direct access to sputum examination but the important privilege of direct free radiographic chest examination and report" (D'Arcy Hart, 1937).

Difficulties Arising During the Survey.

During the whole course of this investigation very few difficulties were encountered. There was the fullest cooperation on the part of the young women and of their employers. In all instances permission was given for the skin testing to be performed on the premises, and this and the subsequent X-ray examination were carried out in the employers' time. One or two firms which were not enthusiastic in 1938 asked to have their staffs investigated in 1939. In some instances, as the result of what was seen of this survey, whole staffs, male and female, were investigated at the request of the employers.

Some space has been devoted to the value of the Mantoux test, because the survey had been in progress only a few days when a medical officer of health questioned the wisdom of going on with it, as "the tuberculin test was of no value in diagnosis". This objection was soon met by pointing out that the skin reaction was used merely as a screen to detect those who had been infected and so were suitable for X-ray examination. No question of the specificity of the test was raised.

Before the work was commenced it was suggested that there might be some opposition from the general practitioners whose patients would be investigated. The Council of the South Australian Branch of the British Medical Association agreed to the survey being undertaken provided that the interests of the general practitioner were adequately protected. The men in general practice showed the greatest interest throughout; they came and inspected the X-ray films of their patients who showed some evidence of tuberculosis, and at the required intervals sent them up for further X-ray examination. Thus a large number of men in private practice were able to watch the regression, and in some cases the progression, of pulmonary tuberculosis. One or two radiologists felt that the X-ray work should not have been done by a physician, and although later several of the subjects were radiologically examined by them, they did not wish to see the earlier X-ray films. In this way, in these few but important instances, the full serial value was lost.

This survey showed the great value of the periodic X-ray examination of people supposedly in good health. The detection of a pulmonary lesion is important; but of far more importance is the demonstration of changes of progression or of regression in these lesions. Several of the young women who had X-ray evidence of pulmonary tuberculosis were either recently married or about to be married. Some had had an ordinary physical examination and were given permission to be married. As a result of this investigation several practitioners said that they would never again give a young woman a certificate of fitness to marry until an X-ray examination had been made.

Economic Security of the Examinees.

In many cases the detection of active tuberculosis and the necessity for giving up their employment caused considerable financial difficulties; often these young women were the bread-winners for the family. The granting of invalid pensions proved of considerable help, for otherwise many would not have been able to continue their treatment. Several firms generously continued to pay their wages for some months or else gave prolonged holidays on reduced pay. In some instances I feel that the period of rest was reduced because the patients were compelled to start earning again. The value of such a survey will be greatly increased when the diagnosis of early pulmonary tuberculosis does not endanger the economic security of the examinees. Finally, in the words of D'Arcy Hart (1937):

> At the first examination after the introduction of a scheme, a number of persons in presumed health have usually been discovered to have serious and open lesions . . . While the discoveries usually provide immediate justification for the scheme as a method of case-finding, it is only after it has been in operation for some little time that its full benefits are experienced, with a fall in the incidence of severe lesions among the active cases discovered.

Summary and Conclusions.

1. In a case-finding survey amongst young women of the age group fifteen to thirty years the Mantoux test was used as a screen to detect those who should have a radiological examination.

2. Of the 3,056 skin tests performed, 1,946 (65%) produced no reaction and 1,110 (35%) produced a reaction.

3. In addition, 216 women aged over thirty years were tested; 53 (24%) gave no reaction and 163 (76%) gave a reaction.

4. The X-ray examination of the 1,110 reactors to the skin test revealed 61 cases of "active or probably active" pulmonary tuberculosis. All these occurred in young women who were supposed to be in good health and were in regular employment.

5. Practically all the lesions were detected in an early stage and all the patients have responded or are responding to treatment.

6. Special attention was paid to the investigation of certain groups, such as nurses, dressmakers, milliners and shop assistants.

7. It was found that cases of early and unsuspected pulmonary tuberculosis did occur among young women in various industries, and these were found in association with the infecting case in these industries.

8. Most cases were detected in the preclinical stage and the sufferers were thus prevented from providing fresh foci of infection.

9. The investigation amongst nurses supported the view that those who did not react to the Mantoux test should not be allowed to nurse patients suffering from pulmonary tuberculosis. In addition, the skin test should be repeated at frequent intervals (three months) until the subject gives a reaction. As soon as this has happened an X-ray examination should be made and repeated three or four times during the first year and once a year for three or four years afterwards.

10. Skin reactions accompanied by vesiculation and necrosis were associated with X-ray evidence of pulmonary infiltration in only one instance.

11. A study of several series of skiagrams supported the opinion that the apical lesion is dangerous and also that a slow blood sedimentation rate is not evidence of inactivity.

12. In the early months of 1938 experiments were performed with microradiography of the image on the fluorescent screen. Dr. Eddy, of the Commonwealth X-Ray and Radium Laboratory, continued to investigate the method. After he had tried various alterations in kilovoltage and exposure time a satisfactory technique was developed. Six young women who were investigated and were found to have early lesions in their lungs were examined by us by means of both microradiography and radiography on the ordinary films, 12 inches by 15 inches in size. It was seen that everything visible on the latter films was plainly visible on the small films when they were projected up to the size of the larger films. The actual cost of the large photograph, developed, was five shillings; the cost of the microradiograph was fourpence, and in large numbers this cost could be considerably reduced. The method of microradiography would be eminently suitable for large surveys. By this method some definite pulmonary tuberculous lesions would be detected at once, and in doubtful cases X-ray photographs on the ordinary sized films could be taken. Thus the expense of future surveys could be materially reduced.

13. An attempt was made to answer the questions why tuberculosis is so common amongst young women belonging to the age group fifteen to thirty years and why the incidence is so high in Adelaide. It is thought that Miss Edna E. Nicholson answered the first question when she stated that "the psychic and physical changes of adolescence and early adult life cause young women to be unusually susceptible to tuberculosis". The general living and working conditions in Adelaide and its suburbs are so good that it is possible that there are a large number of persons with diagnosed and undiagnosed open pulmonary tuberculosis mixing with the general public. It is hoped that a higher standard of sputum examination will do much to lessen this evil.

14. The object of this survey was to find out whether cases of early and unsuspected pulmonary tuberculosis did occur in appreciable numbers amongst young women in various industries. It was able to do this, and to demonstrate that the prognosis is extremely good when the disease is detected in the preclinical stage. But this is only a beginning, and the scheme could be extended to cover a far larger part of the population and be continued for five or ten years. Such a scheme would then be similar to the Prophit Scheme being conducted by the Royal College of Physicians in England. The objects of the latter scheme are as follows:

> . . . to obtain more information about the natural history of tuberculosis; to find out if it is among those who first come in contact with the tubercle bacillus in early adult life, or among those who have already been infected, that fatalities happen; and to decide whether and by what means this can be foretold in individual cases, so that preventive measures may be adopted in time. At the same time the information collected should contribute to the solution of various other problems, including the prognostic significance of the Mantoux test, the interpretation of certain X-ray appearances which are at present of doubtful import, the question of the spontaneous healing of tuberculous lesions even in the absence of treatment, and the diagnosis of cases with latent but progressive disease.

A STUDY OF THE TUBERCULOUS CAVITY OF THE LUNG.[1]

By ROY LE P. MUECKE, M.B., B.S. (Adelaide),
Medical Superintendent, Perth Hospital, Western Australia.

IT has been said that the treatment of pulmonary tuberculosis is the treatment of the tuberculous cavity of the lung. It may be argued that such a conception of treatment is wrong and that the treatment of pulmonary tuberculosis is the prevention of cavity formation. Theoretically this may be so, but in practice it is found only too often that cavitation has occurred by the time the patient seeks medical advice. Furthermore, pulmonary tuberculosis with cavitation has been labelled as "malignant", in contradistinction to the comparatively benign disease of "fibroid" phthisis. One seeks the reason for this emphasis on cavitation in the prognosis of the disease.

Prognosis and Dangers of Cavitation.

In 1936 I analysed the post-mortem findings in 124 consecutive cases of tuberculous cavitation at the London Chest Hospital between the years 1925 and 1936. These were either cases unsuitable for collapse therapy or cases in which collapse therapy was inadequate.

The conclusions, which I summarize below, have been checked by Dr. S. R. Gloyne, pathologist to the London Chest Hospital. These conclusions are as follows:

1. The age period twenty to twenty-nine years furnished the largest group of the series—38·8% (more than twice as many as any other age group).

2. Under the age of twenty years the duration of life from the onset of symptoms was one year; over the age of twenty years it was one and a half to two and a quarter years.

3. Contralateral spread had taken place in 78·2% of cases.

4. The commonest extrapulmonary lesion was tuberculous enteritis, and this occurred just over twice as frequently as tuberculous laryngitis.

5. The majority of cases of tuberculous laryngitis (68%) were associated with ulceration of the intestines.

6. Spontaneous pneumothorax occurred in ten cases, in six following artificial pneumothorax complicated by adhesions.

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on July 17, 1940.

7. Hæmoptysis of any extent was rarely fatal through loss of blood alone, but rather led to terminal bronchopneumonia or acute spread of the lesion.

8. Miliary spread and tuberculous bronchopneumonia occurred in 25·8% of cases.

9. Peritonitis complicating the disease was associated with intestinal ulceration in 79% of cases.

10. Extrapulmonary lesions occurred with much greater frequency in bilateral disease with cavitation than in unilateral disease with cavitation; enteritis was 3·4 times as frequent, laryngitis was 1·5 times as frequent, and spontaneous pneumothorax was 3·4 times as frequent.

11. In unilateral disease with cavitation, 22·2% of subjects died of non-tuberculous bronchopneumonia or pneumonia.

12. Of the associated diseases, it was interesting to find that the incidence of cancer (5%) was the highest in this series. It should be pointed out that only in one case did the cancer exhibit the pathology that is associated with chronic irritation.

During the course of this investigation a number of patients were seen to have survived for five or six years. It is known that a patient with extensive cavitation may survive for long periods under normal conditions of useful life.

In conjunction with these conclusions let us study those compiled by James Watt (King George V Sanatorium)[3] in the estimation of the effect of sanatorium treatment without collapse therapy. Watt attempted to estimate the ultimate result for patients with cavitation. Of the 100 patients traced at the end of five years, 82 had died of their disease, 11 were alive and fit for work, and 7 were alive and unfit for work. The condition of these patients when they were discharged from the sanatorium had been estimated as follows: quiescent, 5 patients; much improved, 25 patients; improved, 26 patients; stationary or worse, 44 patients. Radiographic examination on their admission to the sanatorium had caused the condition of these patients to be classified as follows: recent cavitation, 66 patients; fibrous cavitation, 14; transitional cavitation, 20. Serial radiographs showed the cavitation to have remained unchanged in 50 cases, to have increased in 34 cases, to have decreased in 10 cases, and to have varied in 6. The presumption is, therefore, that 73% of patients with recent cavitation had died.

It is probable that only some 10% to 15% of cavities close spontaneously, despite the extraordinary claims made by Fales and Beaudet,[4] that closure had occurred in 47% of a series of 1,000 cases. These figures have never been substantiated.

Barnes and Barnes,[5] in a study of 616 patients with cavities larger than two centimetres in diameter, found that only 79% survived a year; the average duration of life was 15·8 months.

Schwatt and Rest[6] concluded from their investigation of the cavity bearer that the larger the ulcerating surface, the more ominous the prognosis.

It would appear, therefore, that the prognosis of pulmonary tuberculosis with cavitation and without collapse therapy is comparable with that of malignant disease. With collapse therapy the prognosis is greatly improved. Alexander[5] makes the following statement in his book on collapse therapy:

> The evidence of twenty-five years of experience with collapse therapy strongly favours the assumption that a patient who is discharged as an arrested or apparently arrested case after treatment by collapse therapy has a good, if not considerably better (because of abundant scar tissue formation among the lesions and because of pulmonary immobility) chance of remaining well than a patient similarly classified who was not treated by collapse therapy.

Statistical analyses given and quoted by Alexander leave no loophole for argument. One analysis which indicates the value of collapse therapy is the comparison of the results at the time of discharge obtained by two sanatoria using a wide variety of collapse therapy operations and those obtained by 278 sanatoria using but little collapse therapy. The respective figures for "arrested or apparently arrested" disease were 47·3% and 54·9%, as against 17·0% amongst the sanatoria using little collapse therapy.

Arising out of this short discussion on prognosis and dangers of pulmonary tuberculosis with cavitation are two questions: Why and how do some cavities heal with rest (and sometimes without rest)? What is the mechanism whereby collapse therapy produces healing of the cavity?

Pathology and Pathogenesis.

As the first step in the attempt to comprehend the tuberculous cavity of the lung it is necessary to discuss (even if very briefly) the pathology and pathogenesis of such cavitation.

The tubercle bacillus first gains entrance by ingestion or inhalation, each theory having its protagonists and each individual camp its own divisions of opinion. Furthermore, recent work[6] is producing evidence that the bovine bacillus may also produce cavitation of the lung (that is, adult phthisis).

Whether the bacillus is deposited on the respiratory or alimentary mucous membrane makes very little difference, for the bacillus is non-motile. Its passage takes place passively in the phagocytic cell by which it is engulfed. Within this cell the bacillus traverses the lymph spaces and glands, the thoracic duct and so passes to the blood stream; then it travels through the right side of the heart into the very effective pulmonary filter. Here the phagocytic cell, swollen by pathological and biochemical reactions, becomes arrested in a pulmonary capillary. From this capillary it may once more reach the lymph spaces and continue its travels. Whether it does so or not depends upon the tissue state of the body.

When the development of the disease is considered, two facts should be remembered. First, at birth all individuals are free of tuberculous infection; secondly, all individuals must sooner or later come into contact with the infection.

With the first infection by the bacillus, namely, a first inoculation, certain tissue changes ensue. One of these changes is the ability of the tissues to fix the bacillus and end its nomadism, and the other, an alteration of the sensitivity of the tissue cells so that the body now becomes allergic.

In the reinfection form of tuberculosis, which is almost always seen in the adult, the inoculation of the individual takes place in previously inoculated and therefore sensitive tissue. There is no longer a migration of the bacilli to the regional lymph nodes. They now become fixed as soon as arrested, generally in the pulmonary filter. Accordingly, in the highly sensitive person there is a gross reaction, a low stimulation threshold, while in the relatively insensitive person there is a high stimulation threshold, with the probability of a mild reaction.

The dosage of infection and its interplay with the allergic state decide the clinical picture, which may vary from the acute pneumonic type, with or without tissue destruction, to the comparatively benign disease of fibroid phthisis. The cavity form depends upon the peculiar balance of allergy and dosage which will produce definite lung destruction—epithelioid reaction, giant-cell formation and caseation.

It must be remembered that all the tissues of the lung are affected. Although the pathology is an "alveolus-filling" process, it is not limited simply to the lung parenchyma. Lesions of the bronchi are found, which may be exudative or proliferative in nature, and the formation of a peculiar "granulation tissue", which the French[7] have named "fibro-angiomatous bronchitis" and which, they hold, may maintain persistent sputum not containing tubercle bacilli, despite apparent healing of the lesion. The blood vessels in the diseased areas are found to be the seat of thrombosis, intimal and medial thickening and aneurysmal formation. Finally, with softening and liquefaction of the caseous focus and ulceration into a bronchus or bronchi, the soft caseous matter is expelled into the bronchial tree and a cavity results.

Mechanics and Biology of Cavitation.

The Cavity at Rest.

With the increase in size of the cavity a greater number of bronchi will open into it. It would appear a natural conclusion that increase in size of a cavity is always due

to a progressive tissue destruction. But cavities suddenly stop developing and even decrease in size, disappear and reappear, or may even undergo rapid enlargement so that they inflate like balloons.

In explanation of these phenomena many theories have been advanced. It has been postulated by some that there are two sets of elastic fibres in the lung, one with the property of expansion and the other with the property of retraction. This theory, of course, endows an undifferentiated tissue with properties associated with differentiation. Others attribute the constant enlargement of the cavity to the continuous tension of the elastic tissue. This theory does not explain the many variations that have been observed.

In criticism of these views, Corylios[8] points out that the various authors have disregarded the function of the lung, which is gaseous absorption and gaseous exchange. He lays the foundation of his theory on the final pathological process, which is the very fact of cavity formation, the pathological change in the bronchi. The hole made by rupture into the bronchus and the expulsion of caseous contents might be expected to close, as the elastic tissue of the lung tends always to produce relaxation. This hole, however, finds direct connexion with the atmospheric pressure, which is responsible for the normal distension of the lung and consequently for the spherical shape of the cavity.

Andrus[9] agrees with this view up to a point. His theory is that cavities are probably largely traumatic artefacts of Nature; that they are dilatation of tears in the pulmonary parenchyma subject to the physical forces of the elastic pull of the lung, and therefore with no relation to tissue destruction or gas pressures.

In criticism of this latter conception it should be pointed out that the pathological condition[11] of the cavity wall is characterized by caseation; also that there is a parallelism between the elastic tension of the lung and the balance of intrapulmonary and intrapleural pressures. In his own experiment Andrus has depended upon inflation of the dead lung to make his traumatic tear resolve itself into a cavity.

The corner-stone of the theory advanced by Corylios is the draining bronchus, and with this Andrus agrees. These bronchi are not hypothetical structures, as they can be shown by lipiodol and X-ray examination, by post-mortem and microscopic studies. The view that they should be immune to the tuberculous process is untenable and contrary to fact (for example, Case IV).

There may be found in such bronchi, therefore, caseation with ulceration, stenosis or obstruction, temporary or permanent. In fact, a new growth takes place in the bronchus, which may give rise, just as the bronchogenic carcinoma does, to certain valve mechanisms resulting in atelectasis, emphysema or simply diminished air entry (Cases II and III).

In the case of the bronchogenic carcinoma, it is the lobar or lobular bronchus that is involved. In the case of the tuberculous pulmonary cavity it is the draining bronchus. Instead, therefore, of the lobe or lobule of the lung being affected as in carcinoma of the bronchus, it is the cavity, a single air cell, instead of multiple air cells. Should the draining bronchus become obstructed, atelectasis of the cavity and its related tissue will occur. The cavity will become diminished by absorption of gases, rapidly in young elastic cavities, slowly in large thick-walled cavities, and not at all in the cavity formed by the shelling out of the lung parenchyma of a whole lobe. The sputum will diminish in quantity and will no longer contain tubercle bacilli. Should the bronchus reopen before healing takes place, so will the cavity reappear, together with tubercle bacilli in the sputum.

When a check valve mechanism eventuates, atelectasis or emphysema takes place, and if the latter occurs, there is a rapid increase in the size of the cavity (Case IV).

The cause of the cavity's healing at rest is therefore bronchial obstruction of the type which produces atelectasis. Rest involves, too, a diminution of tidal air and consequently a degree of elastic relaxation. This means, then, the partial elimination of the one antagonist of scar contraction in the lung, namely, elastic tension. Atelectatic shrinkage and fibrous contraction may now take place.

It may be pointed out that the degree of elastic relaxation required in the healing of the tuberculous cavity will vary with the individual. (Compare Cases V, VI, VIII, IX and X.) Moreover, it may not be required at all.

I have watched the disappearance of an apical tuberculous cavity in a woman in the fourth decade, who had refused treatment and who continued to carry out her home duties. The cavity recurred a month after its disappearance, with the return of constitutional symptoms and sputum containing tubercle bacilli.

Collapse Therapy and its Effects.

The next step is to ascertain the relationship of this conception to collapse therapy. Taking the ideal collapse therapy, artificial pneumothorax, as a basis, let me enumerate the changes that follow such collapse in the normal lung. These changes may be divided into three headings:[10]

A. Obvious changes:
- (i) Decreased lung volume.
- (ii) Decreased vital capacity.
- (iii) Increased intrapleural pressure.

B. Changes secondary to *A*:
- (i) Decrease of ventilation (that is, decrease of physiological function and consequently of blood and lymph circulation) if the lung volume is decreased.
- (ii) Partial rest of the lung, but not complete rest, as radiographic and fluoroscopic examination will prove.
- (iii) Kinking and obliteration of minor bronchi.
- (iv) Thickening of the walls of blood vessels.

C. Tissue changes:
- (i) A histiocytic response in septa and in perivascular and peribronchial spaces, with subsequent slight increase in connective tissue.
- (ii) Thickening of pleuræ.
- (iii) Relative local anæmia.

Now let us study the effect of collapse therapy on the tuberculous lung. Primarily, pulmonary tuberculosis impairs the function of the lung parenchyma, in that foci of diseased tissue are unable to function in the hæmorespiratory exchange. Secondarily, further functional disturbance is produced by secondary processes, effusions, adherent pleura, bronchial secretions and constitutional upset.

The general impression of Pinner[10] is that resorption, namely, the absorption of morbid products, is faster in the deflated than in the expanded lung, despite the undeniable fact that circulation is diminished and lymph flow is impeded. It is also recognized that the amount of scarring surrounding a cavity in artificially deflated lungs is much greater than in the lungs in non-surgical cases. An adequate reason for this would surely be the organization of perifocal inflammation, which cannot escape through the obliterated bronchi.

The mechanism of the healing tuberculous cavity under collapse therapy is characterized by one constant accompaniment—elastic relaxation. The one persistent antagonist of contraction is therefore eliminated. Decreased physiological function with a relative anoxæmia and lymph stasis also follows, together with the mechanical closure or kinking of minor bronchi, which produces (or aids in producing) bronchial obstruction. The result of tissue anoxæmia sufficiently pronounced and prolonged is fibrosis.

From these discussions of the tuberculous cavity it would appear that the cycle of events leading to arrest of the disease and cavity healing is the following: (i) the closure of bronchial outlets by pathological processes, by collapse therapy or by both; (ii) arrest of development of toxic products with clinical improvement; (iii) absence of tubercle bacilli from the sputum; (iv) fibrosis of the diseased lung, with contraction of scar tissue and atelectatic shrinkage of pericavital disease; (v) disappearance of cavities.

Many arguments have been brought forward to discredit this theory. Pinner[10] and Auerbach[11] state that cavities present a mechanical difficulty because they probably cannot heal until their walls are brought into apposition. Such a statement would seem to be a ghost rising to greet

us from the days when collapse therapy was compression therapy. Cavities in the expanded lung or in the deflated lung do not heal by approximation of their walls except in the terminal stage of healing. They heal, moreover, by centripetal contraction, a process surely associated with intracavital gas absorption and pericavital contraction (Cases V, VI, VII and IX). The moment effective collapse is attained, the cavity becomes a closed space. A fluid level may therefore appear, or should fluid be present, an increase in the fluid will be seen. Should the pressure within the cavity become increased to an extent that will open the potential draining bronchus or bronchi, there will be a decrease in the fluid level and an apparent increase in the size of the cavity, with the probable recurrence of tubercle bacilli in the sputum (Case VI). When compression therapy, such as high pressure pneumothorax or oleothorax, is used, then the walls of the cavity are approximated at the expense of shape. Should the draining bronchi remain patent, healing will not take place.

The second argument raised is lack of evidence of bronchial stenosis. I have already referred to one case of my own knowledge; Coryllos has reported one with healing of a cavity; Roberts and Pagel[12] reported one case of obstructed bronchus with healing of a cavity. Certain French writers[13] support Coryllos. Post-mortem examinations on the healing cavity are rare, but evidence would seem to be accumulating. Moreover, Coryllos makes no claim that the pathological change occurs in every case, but holds that the effect of collapse adequate to heal cavities depends upon obliteration, with or without pathological change, of the draining bronchi. In the majority of cases of uncomplicated pneumothorax two months or more are required before the sputum ceases to contain tubercle bacilli, the process of closure of bronchi being as gradual as the progress of collapse. This fact is to be contrasted with the almost immediate disappearance of tubercle bacilli from the sputum following complete section of adhesions in an established artificial pneumothorax,[14] an observation which I believe has only one interpretation—obliteration of the draining bronchi.

Collapse does not mean atelectasis or the anoxæmia comparable with that produced in atelectasis. I have already, in this theory, attempted to show that selective atelectasis does occur, with atelectatic fibrous shrinking or pericavital disease. That the aerobic tubercle bacillus may still obtain its oxygen by means of the circulation is doubtful. Post-mortem examinations have shown the blood vessels in the diseased areas to be frequently thrombosed, or the seat of intimal and medial thickening. This thickening is further increased by the effect of collapse; therefore the artery is left still less capable of functional activity.

Experience would tend to prove that the danger of bronchial stenosis is largely theoretical. It must be remembered that the pyogenic organisms are secondary in tuberculous cavitation, and not primary, as in the case of lung abscess,[15] upon which corollary the danger of bronchial stenosis is based. In the tuberculous cavity the secondary invader falls upon a soil devitalized by the tuberculous process and rendered resistant by a wall of fibrous tissue with caseous or retrogressing caseous areas. This supposition is further enhanced by the comparative lack of fœtid or offensive sputum in the case of tuberculous cavitation.

Summary.

This conception establishes the healing action of rest in bed in the more acute types of cavity by the diminution of tidal air. Tension in the lung is thus diminished. This helps in the obliteration of bronchi affected by the same exudative process. Rest in bed decreases pulmonary ventilation, and this in its turn leads to a limitation of oxygen (necessary for life and growth of the tubercle bacillus) in a tissue in which pathological changes in blood vessels are already causing a limitation in hæmorespiratory exchange.

It establishes, too, the similarity of action of artificial pneumothorax, thoracoplasty, phrenic paralysis (Case X) *et cetera* in their facilitation of healing of tuberculous cavities. They are surgical measures which depend upon decrease of lung volume (that is, elastic relaxation), suppression of ventilation and diminution of tidal air, so that the draining bronchi may be aided actively in their closure and that scar contraction may be encouraged.

The old argument as to the relative value of phrenic paralysis in the treatment of apical and basal cavities is decided once and for all. So long as the phrenic paralysis can procure for the lung so affected a sufficient diminution in lung volume and therefore elastic relaxation in relation to the cavities and draining bronchi present, then those cavities will heal regardless of their position in the lung.

Such a conception gives reason for the trend of modern treatment, namely, in the following measures: (i) the increasing use of artificial pneumothorax and, when this fails, the resort to major surgical procedures, such as thoracoplasty; (ii) the introduction of section of adhesions, by both open and closed methods; (iii) the development of the technique of thoracoplasty to achieve, as far as possible, the effect produced by artificial pneumothorax;[13][14] (iv) a decrease of those measures depending upon high intrapleural pressures.

Today tuberculous cavitation of the lung depends upon surgery for its adequate treatment, from the comparatively simple induction of artificial pneumothorax to major surgical procedures, such as thoracoplasty. Admittedly, cavitation will yield to rest in bed; but the risk of a recurrence is definite. This has been more than an occasional observation in a short experience.

In view of the dangers to which the cavity bearer is subject (and of those to which he subjects others), it is unwise to delay collapse therapy when the condition lends itself to such therapy. The failure of artificial pneumothorax should mean the immediate consideration of the larger surgical procedures involving discussion by physician, surgeon and anæsthetist. I do not contend that collapse therapy should displace the sanatorium from the armamentarium of treatment, but that collapse therapy and the sanatorium should go hand in hand; that the sanatorium should be the centre for active treatment as well as for conservative treatment.

Pulmonary tuberculosis with cavitation assumes the proportions of a malignant disease. Therefore, if operation is possible, it should be performed without undue delay. As far back as 1898 John B. Murphy made the statement that tuberculosis of the lungs was yielding to surgical principles, and this statement finds a louder echo in each year that passes.

Reports of Cases.

CASE I.—Figures Ia, Ib and Ic illustrate the behaviour of tuberculous cavitation in a female patient, aged twenty-eight years. This patient had symptoms and conclusive signs of pulmonary tuberculosis. In the course of seven months there occurred radiologically visualized disappearance of the

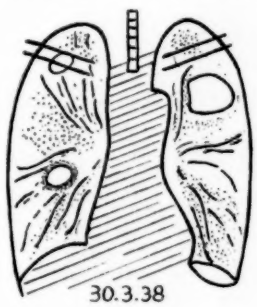

FIGURE Ia.

cavities in the right lung simultaneously with growth of the existing cavity and the formation of new cavities in the left lung.

A curious anomaly thus presented itself, namely, the radiological healing of tuberculous cavitation in one lung,

despite progression of the disease, clinically and radiologically observed progress in the other lung. The conception of the tuberculous cavity explains this anomaly.

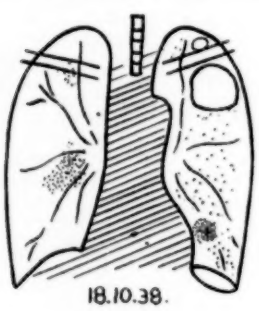

FIGURE IB.

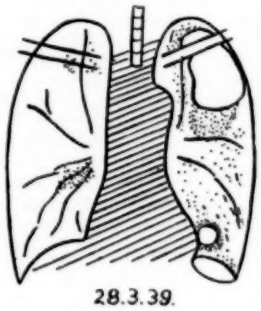

FIGURE IC.

Cases II and III demonstrate types of the valve action mechanisms produced by bronchial newgrowths.

CASES II and III.—The radiograph in Case II showed collapse of the lower lobe of the right lung (Figure IIA). A bronchoscopic examination proved the presence of a simple polypus, situated as shown in Figure IIB. This polypus was impacted in the lower lobe bronchus of the right lung, so that there was no air entry into the lower lobe. The result was atelectasis, and, secondarily, infection and bronchiectasis.

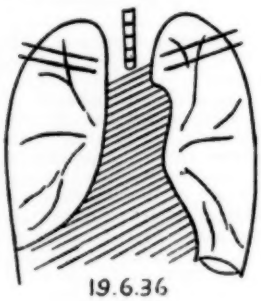

FIGURE IIA.

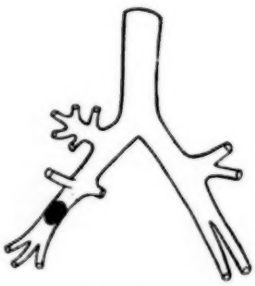

FIGURE IIB.

The radiograph in Case III revealed an obstructive emphysema (Figure IIIA), and bronchoscopic examination revealed the presence of a simple polypus situated as shown in Figure IIIB. This polypus acted as a bivalve mechanism in relation to the lower lobe of the left lung, giving, on examination of the patient, signs of diminished air entry. In relation to the upper lobe of the left lung it acted as a check valve, and on inspiration an airway to the lobe

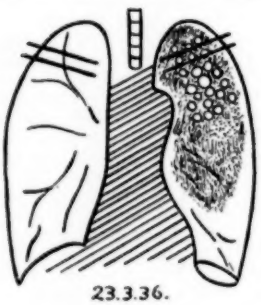

FIGURE IIIA.

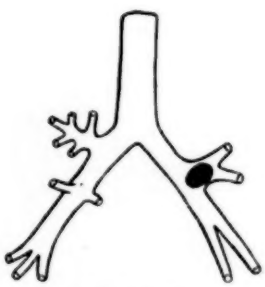

FIGURE IIIB.

could be seen to form. However, on expiration this space became closed immediately elastic relaxation of the lung occurred, so that the air inspired by the upper lobe of the left lung was trapped in that lobe. This patient suffered from three attacks of "left apical pneumonia" within six months. On the third attack he was admitted to the London Chest Hospital. After the removal of the polypus his symptoms quickly subsided and his convalescence was uneventful. A subsequent bronchographic examination provided no evidence of bronchiectasis in the upper lobe of the left lung.

CASE IV.—Case IV has also been reported by Dr. Morlock;[19] it is the case of a girl, aged eighteen years, suffering from pulmonary tuberculosis with cavitation. She was admitted to the London Chest Hospital in January, 1936. In February, the condition being as shown in Figure IVA, an artificial pneumothorax was induced on the left side, and in March the adhesions were severed (Figure IVB). Despite this, the cavity rapidly increased in size, the mediastinum became progressively displaced to the right, and estimations of intrapleural pressure gave higher and higher figures. The presence of fluid covering the diaphragm did not suffice to account for these findings.

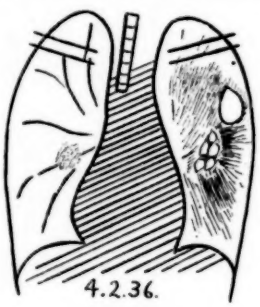

FIGURE IVA.

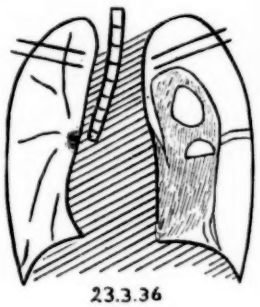

FIGURE IVB.

In May Dr. Morlock examined the patient bronchoscopically and the lower lobe bronchus was found to be the site of granulation tissue, which obstructed the lumen of the bronchus completely on expiration (Figures IVC and IVD). On inspiration, with widening of the bronchus, an airway was effected to the lower lobe.

This abnormal condition, combined with collapse therapy, had failed to obliterate the draining bronchi, but had produced an effective valve mechanism in the lower lobe bronchus. Each inspiration, therefore, contributed towards the ballooning of the cavity and lower lobe tissues. There was no escape on expiration. When cauterization and dilatation of this granulation tissue and stenosed bronchus were carried out there followed a return to normal, a decrease in the size of the cavity *et cetera*. However, the condition recurred.

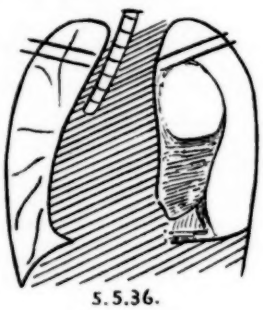

FIGURE IVC.

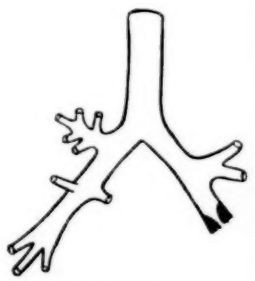

FIGURE IVD.

This case demonstrates a definite bronchial abnormality secondary to tuberculosis, contributing towards cavity enlargement by the production of a valve mechanism.

Case V demonstrates the growth of tuberculous cavitation with adhesions preventing an effective pneumothorax.

CASE V.—The cavity on April 22, 1938, was 3·5 centimetres by 3·0 centimetres in area (Figure VA); on May 25, 1938, its area was 3·5 centimetres by 2·0 centimetres (Figure VB). The patient refused internal pneumolysis. His sputum continued to contain tubercle bacilli; he continued to suffer from definite malaise, intermittent pyrexia and loss of weight. Because of his continued ill health he eventually decided to undergo the operation advised. By this time, on September 13, 1939, the cavity was 4·5 centimetres by 3·6 centimetres in area (Figures VC and VD). One month

after section of the adhesions the cavity measured 1·0 centimetre in diameter (Figure VE); two months after, it had disappeared.

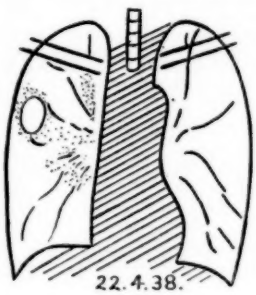

FIGURE VA.

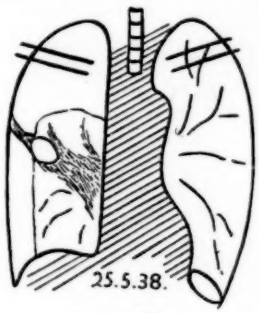

FIGURE VB.

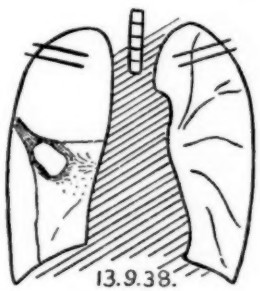

FIGURE VC.

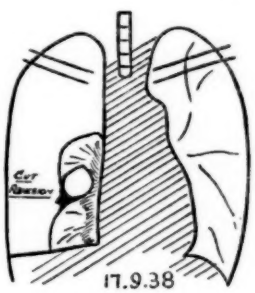

FIGURE VD.

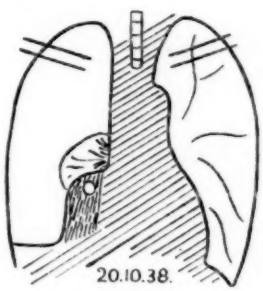

FIGURE VE.

Despite artificial pneumothorax, there was a definite increase in the size of the cavity, probably due, in view of the constitutional symptoms, to actual activity of the disease process. Undoubtedly the complicating adhesion was responsible for the failure of the cavity to heal.

It was interesting to note, once complete relaxation of the lung was obtained, the rapid response of the cavity to collapse therapy. Immediate disappearance from the sputum of tubercle bacilli which had previously been present, disappearance of constitutional symptoms and the centripetal healing of the cavity all followed. This sequence of events provides strong evidence of obliteration of the draining bronchi.

The figures in the following case are from the X-ray films of a patient treated by artificial pneumothorax, internal pneumolysis and thoracoplasty. To my mind they ably demonstrate the part played by the draining bronchi in the healing of tuberculous cavities in the lung.

CASE VI.—The original cavity was approximately 4·3 centimetres in diameter (Figure VIA). The pneumothorax was complicated by adhesions, and the cavity on February 18, 1938, measured some 5·0 centimetres by 3·2 centimetres (Figure VIB). On February 25, 1938, internal pneumolysis was performed, but complete section of adhesions was not possible.

In the four months that followed, the sequence of events shown by Figures VIC and VID, from the X-ray films taken on May 4, 1938, and on June 1, 1938, occurred on three occasions. First there was the gradual increase of fluid level, which decreased the size of the cavity to an area 2·5 centimetres by 2·0 centimetres, and during this phase examination of the sputum failed to reveal the tubercle bacillus. This was followed by a sudden decrease of fluid, an increase in the size of the cavity to an area of 5·0 centimetres by 2·5 centimetres, and the sputum contained tubercle bacilli.

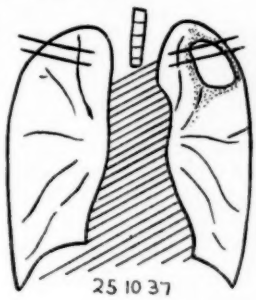

FIGURE VIA.

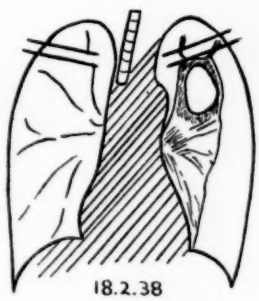

FIGURE VIB.

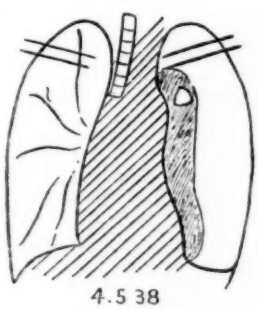

FIGURE VIC.

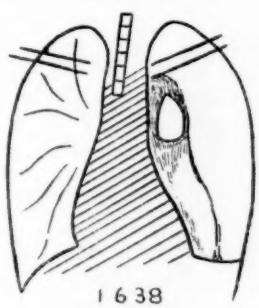

FIGURE VID.

The patient was then referred for thoracoplasty. In retrospect I firmly believe that we were somewhat precipitate in the advising of such a major surgical procedure, as phrenic paralysis might well have achieved our aims. However, thoracoplasty was performed according to the Semb technique; the result as shown in Figures VIE and VIF represents the condition on November 8, 1938, and on December 3, 1938.

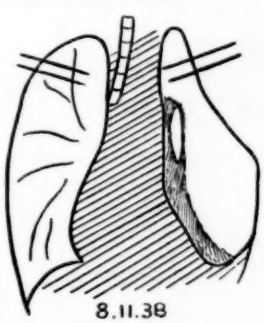

FIGURE VIE.

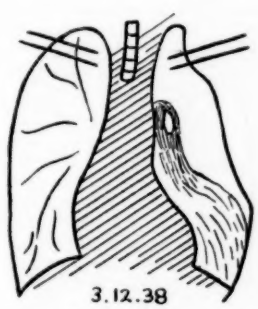

FIGURE VIF.

In this case pneumothorax was complicated by adhesions which could not be completely severed. That portion of adhesion remaining was sufficient to prevent healing of the cavity. Despite the fact that fluid periodically collected within the cavity, the increasing intracavital pressure was sufficient to open the potential draining bronchi and so to establish drainage with the reappearance of tubercle bacilli in the sputum. With the performance of thoracoplasty there was achieved not only

compression, but also further mobilization of the apex. As a result of compression the cavity suffered at the expense of shape and not of volume (according to measurements). There then occurred a fluid level in the cavity and finally radiological disappearance; meanwhile the sputum remained persistently free from tubercle bacilli; that is to say, the draining bronchi were closed.

CASE VII.—Figures VIIA, VIIB and VIIC demonstrate the behaviour of a tuberculous cavity after an effective thoracoplasty with extrapleural apicolysis. Again, there is not the approximation of walls, but rather the centripetal diminution of the cavity.

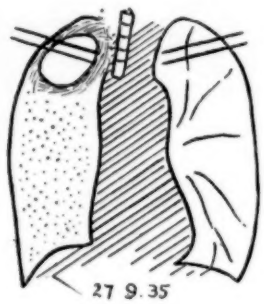

FIGURE VIIA.

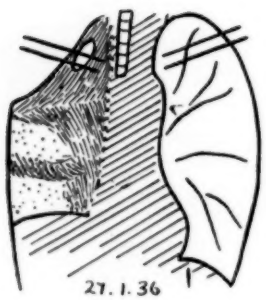

FIGURE VIIB.

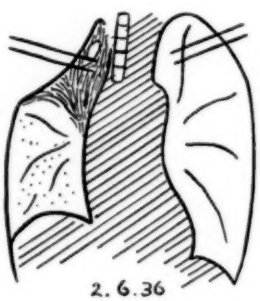

FIGURE VIIC.

CASE VIII.—Case VIII is the history of a patient suffering from pulmonary tuberculosis (Figure VIIIA), who was admitted to hospital with recurrent hæmoptyses. After induction of the pneumothorax, cavitation became obvious (Figure VIIIB). Despite a pneumothorax which appeared radiologically to be uncomplicated (the patient was not examined by means of thoracoscopy), progressive enlargement of the cavity was seen, from 1·9 centimetres in diameter to 2·2 centimetres in diameter on September 26, 1938 (Figure VIIIC), and to 3·2 centimetres in diameter on December 2, 1938 (Figure VIIID). The daily quantity of sputum was three to four ounces, and the sputum was constantly loaded with tubercle bacilli. The patient's physical condition gradually deteriorated.

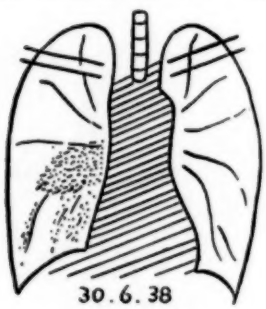

FIGURE VIIIA.

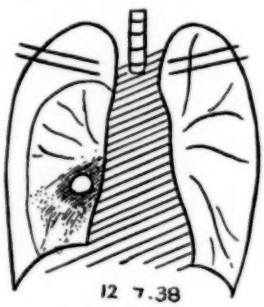

FIGURE VIIIB.

Bearing in mind Case IV, I decided to examine the patient by means of bronchoscopy. The bronchial tree, except for the right middle lobe bronchus, was dry and normal, except for some general congestion of the mucosa, particularly of the right stem bronchus. The opening of the right middle lobe bronchus was swollen and somewhat distorted, and there was a persistent discharge from this bronchus, mucopus being expelled with expiration and partly inhaled back into the bronchus again on inspiration. Therefore, directly or indirectly, the middle lobe bronchus was draining the cavity. Bronschoscopic examination proved this drainage to be patent. The cavity increased in size.

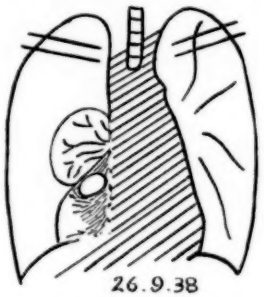

FIGURE VIIIC.

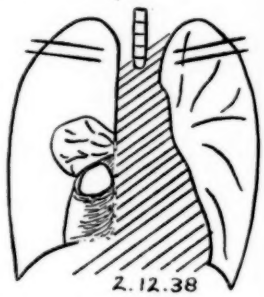

FIGURE VIIID.

The patient was discharged to a sanatorium, where larger refills and higher pressures were instituted. The sputum decreased in quantity, the general condition improved and the cavity healed.

CASE IX.—Figures IXA and IXB demonstrate shrinkage of a cavity in a patient who was treated simply with rest in bed. The cavity was typically the "annular" shadow with a "cotton-wool" surrounding, namely, the subacute cavity. In one month the size of the cavity had decreased from 4·2 centimetres in diameter to 2·2 centimetres in diameter. Within three months there was complete radiological disappearance.

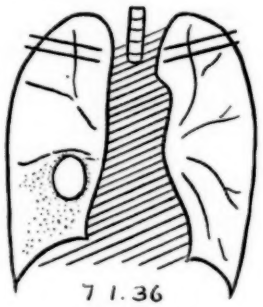

FIGURE IXA.

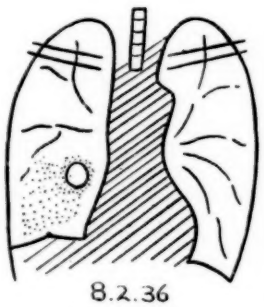

FIGURE IXB.

CASE X.—Case X illustrates the effect of phrenic avulsion in a male, aged thirty-three years, who was in 1934 treated by artificial pneumothorax for right apical tuberculosis with cavitation. There were adhesions complicating the pneumothorax. In September, 1936, after an apparent cure (the resultant scarring is shown schematically), there was

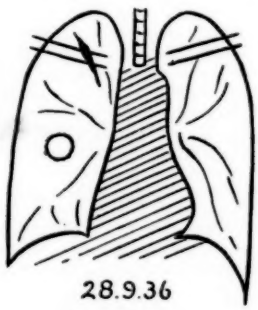

FIGURE XA.

a return of symptoms, and an X-ray examination on September 28, 1936, revealed the presence of a new cavity (Figure XA). In five weeks, despite rest in bed, the cavity had doubled its size and its walls had become thicker (Figure XB). A phrenic avulsion was performed, reinduction of an artificial pneumothorax proving unsuccessful. Within five weeks of the operation (December 18, 1936) the cavity had disappeared and the patient was well (Figure XC). An X-ray film taken in January, 1940, showed that the lungs had remained free from cavitation during the three succeeding years.

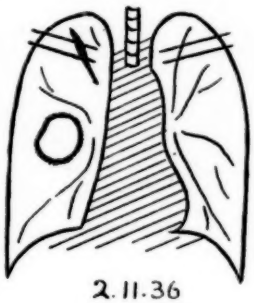

FIGURE XB.

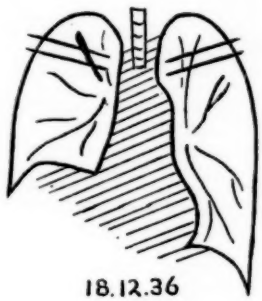

FIGURE XC.

Acknowledgement.

I wish to acknowledge my indebtedness to Dr. S. R. Gloyne and Dr. H. V. Morlock, of the London Chest Hospital; to Dr. J. G. Hislop, senior physician of the Perth Hospital, and to Dr. B. A. Hunt, for their encouragement and help; and to many members of the honorary medical staff of this same hospital for permission of access to their patients. I should also thank very sincerely Miss M. Hodgson for her assistance in the schematic diagrams which are here reproduced.

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TUBERCULOSIS AS A PROBLEM FOR THE STATE.[1]

By COTTER HARVEY,
Sydney.

"THE first consideration of a State should be the health of its people." This dictum of the famous British statesman Lord Beaconsfield, almost a translation of the Roman law *"Salus populi, suprema lex"*, has been appreciated only in recent years. It is evident throughout the world that the State is assuming ever-widening responsibilities in regard to public health and the prevention of disease, and this is especially true in regard to tuberculosis. An enlightened community has the right to demand a proper organization and coordination of effort by the State against tuberculosis. That there should be a special organization functioning for the control of this disease is no longer a subject for debate.

My next point is to urge that after an evaluation has been made of the tuberculosis problem as it concerns your particular State, a comprehensive programme should be drawn up. I have no doubt this has been done ere this, as it will be obvious to you all that the success of any anti-tuberculosis campaign must be governed by the degree of completeness and efficiency of the plan which is adopted. This, in the words of Sir Robert Philp, the renowned Scottish pioneer of anti-tuberculosis measures, who started the first tuberculosis dispensary in 1887, demands "the harmonious co-ordination of well-directed measures". Piecemeal efforts, I would warn you, are largely futile and always uneconomic. Of what use, for instance, is it to establish diagnostic facilities for finding patients with early lesions if no beds are available for their treatment?

By this I do not intend to imply that any scheme can be put into operation all at once. But there must be an orderly and carefully thought out sequence of events—in short, a real programme. To the instance I have given above the correct reply is obviously: first, provide the necessary accommodation, then initiate a case-finding campaign. Dr. M. J. Holmes, in his report to the eighth session of the National Health and Medical Research Council (May, 1940), offers a trenchant criticism of this problem:

> It would be a tragedy and a damaging reflection on any administration to find the cases only to advise them that for lack of accommodation they must wait their turn for treatment under suitable conditions.

Let me now offer you two simple postulates, which are fundamental to our appreciation of the problem we are about to face: (i) tuberculosis is an infective condition, (ii) it is in large part the expression of faulty social conditions. On these two facts rests the most important aspect of our campaign, that of prevention. This is the ideal towards which we must strive, and to which all our efforts, though apparently in other directions, actually tend in the long run. "An ounce of prevention is worth a ton of cure"; the truth of this was never better exemplified than in tuberculosis.

Prevention may be considered under four headings: (i) control of patients with "open" lesions, (ii) systematic examination of contacts, (iii) community surveys, (iv) increasing of resistance, racial and individual. A full discussion on this aspect could well occupy all my available time. I can touch on only a few points:

(i) Ideally all the people who have tubercle bacilli in their sputum should be isolated. There are many obstacles in the way of a practical realization of this ideal; but perhaps we are not so far off as some people think. Remember meanwhile that the patient with tubercle bacilli in the sputum, particularly in Queensland, where bovine tuberculosis is practically unknown, is the root of the whole evil. How he is to be discovered and dealt with may become apparent later.

(ii) The thorough examination of all contacts, old and young, family or remote, is essential. In a well-organized

[1] A post-graduate lecture delivered in Brisbane on May 30, 1940.

scheme this is interpreted widely. (For example, when a school teacher is tuberculous, all the children he has taught should be examined; when a workman is tuberculous, all his close associates at work.) The examination includes a skin test (Mantoux) and the taking of an X-ray film if there is a reaction to the skin test, and child contacts should, if free from tuberculosis on the first visit, report at regular intervals, at least until they are grown up.

(iii) The most important advance in preventive medicine of this century promises to be the survey of groups of persons or even of whole communities by miniature X-ray photography, or, as it is now known, photo-fluorography of the chest. By this means tuberculosis is discovered before the individual can be made aware of it. As you no doubt all know, this method has revealed pulmonary tuberculosis in approximately 0·5% of men who had passed medical examinations for the Australian Imperial Force, and it is fair to estimate that at least 2% of the adult population is so affected. The following are the results of a recent survey made in Sydney by photo-fluorography, under the auspices of the New South Wales Department of Public Health (Tuberculosis Division). In all, over a period of approximately six months (1939-1940), 672 persons were examined in 11 groups. It will be seen that the percentage of affected persons is far higher than that found by medical examinations of recruits.

Evidence of pulmonary tuberculosis ..	62 (9%)
Lesions regarded as active	34 (5%)
Lesions regarded as healed	28 (4%)

But the people examined, though apparently in good health, were mainly contacts, and thus did not constitute a random sample of the population of the city.

Let me clarify one point. A mass survey such as this does not pretend to offer a final diagnosis. Its object is to select for more careful study a relatively small number of persons from a large population of presumably healthy individuals. Short of the entire population, selected groups can be taken, such as contacts, nurses, high school students, men and women in industry, or women having prenatal care. Dr. Wunderly, of Adelaide, who has gone carefully into this question, and to whom I am indebted for much information on the subject, estimates that in a mass survey the over-all cost of a Mantoux test and a photo-fluorograph would be one shilling per head. As against this, it has been stated that the average cost to the State is £1,000 for each tuberculous patient. The Repatriation Department, more generous in its treatment of pensioners, has let it be known that each tuberculous soldier from the last war has cost them £3,000. Just ponder over the difference between these figures and then ask yourself how far prevention is worth while, on a purely economic basis.

(iv) Lastly we come to the problems arising from our imperfect civilization: the social and economic factors. Overcrowding and undernourishment are powerful allies of the tubercle bacillus. Attempts must be made to improve the standard of living of the poorer people, and an adequate diet must be ensured for all. There is room for much thoughtful endeavour in these directions, and Queensland may here take pride in leading the way among the Australian States, with its enlightened propaganda on nutrition.

In addition to prevention there are four other lines along which the State must proceed. These are summarized as follows:

State administration of tuberculosis:

(i) Prevention.
(ii) Diagnosis.
(iii) Treatment.
(iv) After-care and rehabilitation.
(v) Teaching and research.

Adequate facilities must be provided for diagnosis and for treatment in all stages, by means of all modern methods, both medical and surgical. I refer here particularly to the various forms of collapse therapy, the usefulness of which is not yet sufficiently appreciated.

There is next an important problem which in Australia has hardly been touched on. What is to be done for the patient after he has passed through his period of treatment? Some can achieve their main object, which is to return to their former occupation, while many become complete invalids and need institutional treatment for the remainder of their days. A large group, however, is left "substandard", able to perform some work, but unable to compete in the labour market. For economic reasons, and for the sake of the patient's morale (life without purpose is life without value), we must recognize this group. They now are either content to eke out a precarious existence by means of the invalid pension, or more ambitious, more conscious of their responsibilities, return to work, break down in health and die, leaving their family a burden on the State. For a proportion of the tuberculous, therefore, we must provide means either for gradual and complete rehabilitation (namely, a return to a normal working wage) or for permanent after-care with partial employment, and thus a purpose in life. It is necessary that the State shall support after-care in the realization that it is an essential part of treatment, as important a factor really in the tuberculosis problem as any clinical measures.

Lastly, I come to the need for provision in the scheme for adequate teaching of the many-sided problems of tuberculosis—medical, social, economic, and so on. Students and nurses should receive more training than they do now, and post-graduate classes should be held regularly. I fear that the average general practitioner has but a sketchy knowledge of a subject in which tremendous advances have been made in recent years. With teaching, of course, goes research, a wide field for which exists, with many unanswered problems awaiting solution.

Let us now consider the rough scheme I have drawn up as a model for tuberculosis control (Figure I). For simplicity and reasons of time it is incomplete. Preventoria are not mentioned, for instance, nor is the economic aspect, nor that of propaganda.

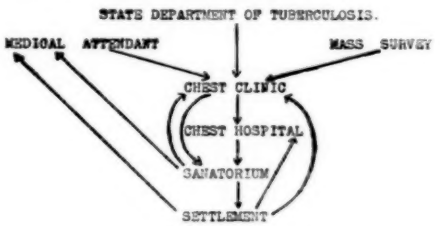

FIGURE I.
Scheme for tuberculosis control.

I would urge the desirability of the creation of a special department of tuberculosis, under a whole-time director who has had wide experience in tuberculosis (this is of more importance, in my opinion, than a diploma of public health) and in whom is vested some authority. His duties I shall not attempt to define, but his position will be no sinecure; he will have to conserve public health prerogatives, while at the same time avoiding any infringement of the rights of the private practitioner.

At first the patients will be found by the general practitioners and out-patient departments of the public hospitals. Later, by propaganda, other persons than the sick, notably contacts, will present themselves for medical examination. But the real "catch" of the future, the early, preclinical, "hopeful" cases, will be "netted" in those group and community surveys to which I have already referred.

And now we come to the backbone of any scheme, the tuberculosis dispensary, more simply and euphemistically called the chest clinic. It is the diagnostic clearing house, the centre for contact and other group examinations, for ambulatory treatment by artificial pneumothorax and for periodic review of those who have had other treatment. By its visiting sisters it undertakes to teach hygiene in the home, to round up the contacts, to follow up the patients, and to provide a liaison between general practitioners and clinic doctors. All these and many other duties it performs, consultative and social services being outstanding.

I am convinced that the clinic is best attached to a general hospital and not run as a separate unit. By this means all ancillary services are available to it, and it is more economical to run. Also the stigma of tuberculosis can be avoided and patients are not deterred from coming for treatment. The figures of the Royal North Shore Hospital out-patient clinic of the pulmonary unit show how closely linked is the clinic with the hospital. Of two hundred consecutive patients, 100 male and 100 female, a certain number were dealt with as follows:

Admitted to hospital for investigation or treatment	58 (29%)
Referred to other out-patient departments ..	64 (32%)
Sent to clinic from hospital	16 (8%)

A recognized standard is to establish one clinic for each 250,000 inhabitants. I would make bold to suggest that one clinic be established in Brisbane and that in addition a travelling clinic be set up to visit other cities and country centres. The cost would not be great, and I believe the results would be worth while.

A modern but essential unit in our scheme is the hospital. Three main functions can be allotted to it: first, to treat on modern lines both medical and surgical patients in various stages of the disease, in special medical and surgical wards. Secondly, to give initial treatment to patients who need complete rest preparatory to sanatorium treatment. At present patients with an acute onset (and they form a considerable percentage) are excluded from sanatoria when they urgently need rest in bed. There are, in fact, far too many tuberculous patients walking about who should be in bed. Further, there is great preventive importance in sending a patient to hospital immediately his condition is diagnosed. Thirdly, the hospital should tend those patients with advanced disease who are not likely to recover.

The question now arises, how many beds should the hospital afford? If the standard of two institutional beds for every tuberculosis death is accepted, Queensland should have approximately 600 beds for its tuberculous patients. I suggest, with deference, that 100 of these should be in a special tuberculosis ward of the general hospital.

The next link is the well-tried one, that of the sanatorium. Rest is now regarded as being more important than climate in treatment, and accessibility to main population centres takes precedence over altitude in the determination of a sanatorium site. There should be more than one sanatorium in Queensland, in view of its widely scattered population, and at least 300 beds should be available. A sanatorium of at least 200 beds to serve the city of Brisbane should be built immediately, and following modern world trends, it should be well within a radius of ten miles from the civic centre. More than this I cannot say, except to emphasize that a sanatorium of less than 100 beds is generally regarded as uneconomic, and that one medical officer should be provided for every 50 patients.

The village settlement, known to you all through Papworth and Varrier Jones, is the best answer yet provided to the problem of after-care. I regret I have not time to discuss this further.

It is essential to realize at this stage that the State must not only provide the links; it must also make it economically possible for the patient to go through them. The economic factor is of vital importance, and the invalid pension is certainly not adequate for the patient and his family while he is undergoing treatment which in the average case should necessitate at least two years away from work. I take the liberty of quoting Dr. Holmes again:

> No scheme for the control of tuberculosis can be effective unless the economic factors receive adequate recognition and are covered by some system for ensuring suitable environment and nutrition for the dependants of the tuberculous patient.

Here again is a subject that might be discussed at length.

I regret that I have left many facets untouched, such as legislative measures, preventoria and special schools, and the interrelation of Federal and State Governments, and I am aware that I have dealt inadequately with some of the most important aspects. The problem is a vast and many-sided one, and there is room for much difference of opinion regarding the best solution. I have attempted to offer you the framework of a plan, based on my experience and on observations made while abroad. If adopted in your State this scheme could be made to work and, more important, to pay big dividends both in lives and in money.

It may be asked, why bother about such minor problems as tuberculosis when the country is at war, in a life and death struggle? Or it may be said that there is no money now for any such schemes as I have described. To those critics I would reply in Sir Robert Philp's words of twenty-two years ago: "The stern events of war throw into starker relief the urgent need for a wisely directed comprehensive campaign against tuberculosis." It is almost certain that not only in the devastated countries, but throughout the world, the incidence of and mortality from this disease will rise. Prolonged physical and mental strain will be the lot of most, if not all, of us; overcrowding and undernutrition must increase as we tighten our economic belt. In the post-war period there will be a harvest of tuberculosis for which we must not be unprepared. Human lives are going to be more precious than ever before, and we cannot afford to lose a single one from any preventible cause.

Let us, then, see to it that our house is set in order and that an efficient tuberculosis service is built up forthwith, not only in Queensland, but throughout the Commonwealth.

Acknowledgement.

I am indebted to Dr. John Hughes, of the Tuberculosis Division of the Department of Public Health, New South Wales, for the figures relating to the photo-fluorographic survey.

Reports of Cases.

MELANOTIC SARCOMA IN A NATIVE OF NEW BRITAIN.

By A. V. G. PRICE,

Government Medical Officer, Kieta, Territory of New Guinea.

THE following case is of interest on account of the occurrence of a melanotic sarcoma in a Melanesian native. Iatola, a male native of the Kokopo district of New Britain, whose age was estimated at about fifty years, was admitted to the Native Hospital on October 24, 1934, with a heaped-up granulating mass on the sole of the left foot. This was considered to be due to yaws or to an infection around a foreign body, or to be possibly a newgrowth. There were some firm enlarged glands in the left groin. His state of nutrition was fair, but not good, and his general health was of the usual standard for a native eating a village diet and for his apparent age. Examination of the chest and abdomen revealed nothing to indicate the presence of secondary deposits of a newgrowth. He was given two injections of "Novarsenobillon", separated by an interval of a week; this is usually sufficient to cause an acute lesion of yaws to show signs of receding. Bluestone was applied locally and the lesion was dressed with glycerine and magnesium sulphate paste. The patient was given a potassium iodide mixture.

Two weeks later the lesion was larger and fungating. The patient was given a third injection of "Novarsenobillon" and the mass was excised, as far as could be judged, beyond the limits of the growth. The cavity was left to heal by granulation.

The mass, on being cut across, was of firm consistency and in patches darkly pigmented; it was thought to be possibly a melanotic sarcoma. It was sent to Dr. T. C. Backhouse, Government Pathologist at Rabaul, who reported a week later that the growth was probably a melanotic sarcoma.

On November 15, 1934, the enlarged glands were removed from the left groin. Dr. H. C. Hosking, Government Radiologist at Rabaul, examined skiagrams of the skull, thorax,

pelvis and left foot, and reported that there was no definite evidence of metastases in these regions. Dr. Backhouse reported on the tumour and glands as follows:

> Macroscopic: Section shows masses of tumour tissue, both non-pigmented and highly pigmented, invading the subcutaneous fat. Limit of gross invasion appears to have just been included in excision.
>
> Microscopic: Non-pigmented portion shows masses of very irregular cells having a roughly alveolar arrangement. Some pigment-bearing cells are present. The grossly pigmented portion of the growth shows masses of cells tending to be spindle-shaped and mostly highly pigmented. Many masses of extracellular pigment present. There is a dense round-cell reaction in the rather scanty supporting stroma.
>
> Melanoblastoma, clinically, melanotic sarcoma; appears highly malignant.
>
> Groin gland, microscopic: Gland extensively invaded and almost replaced by irregular ovoid and spindle cells and masses of pigment. Secondary melanoblastoma of lymph gland.

Dr. A. H. Tebbutt, of Sydney, kindly examined the sections at the request of Dr. Backhouse and reported a fairly typical melanoma with carcinomatous structure.

The wound filled in, leaving a flat ulcer, which became covered with epithelium and had healed by February 19, 1935. The wound in the thigh healed without any trouble. The patient was looking well and had gained in weight with improved diet under hospital conditions. A second series of X-ray photographs taken on April 1, 1935, was examined by me in Dr. Hosking's absence from the Territory of New Guinea. I could see nothing new in them, and no definite evidence of metastases. The patient was discharged from hospital on April 25, 1935, apparently well, and arrangements were made for his return at intervals for inspection. As I had left for another district, Dr. Backhouse saw him from time to time. He supplied information that the patient was apparently free from recurrence until the inspection at the beginning of October, 1935, when there was evidence of a recurrence in the scar on the foot, described as "a small dark-looking patch over which the epidermis has broken, and lateral to this a bulging of the same".

In January, 1936, I had an opportunity of examining the patient again. He was fat and looked well. There were some induration about the scar and a mass extending beyond it for an inch or so under the skin and causing it to bulge. This was undoubtedly a local recurrence. No enlarged glands were palpable in the groin and there was no clinical evidence of secondary deposits in other parts. The subsequent history of the patient is unknown.

Discussion.

This is, as far as I am aware, the first published instance of a melanotic sarcoma in a native of this region, and it is evidently an uncommon condition, as Dr. Backhouse informs me that in laboratory records dating back to 1922 he has notes of only one other such case, excluding a less well-defined tumour of the eye. This was a case in a native of Admiralty Island, from whom material was sent to Dr. G. M. Heydon by Dr. S. J. Cantor in April, 1924. Dr. Heydon's report reads: "Soft tumour about the size of a golf ball, piebald black and cream colour. Microscopically: melanotic sarcoma, many cells fusiform in shape." Dr. Cantor reported three months later that the growth, which had been excised from the foot of an elderly male native, had recurred in fungoid form and that progressive loss of weight had taken place.

Very little has been published in regard to malignant disease in natives of New Guinea. In 1933 Deland, Bull and Cleland[1] reported a rodent ulcer in a native of Bougainville, and Penington[2] in the same year reported on twelve tumours in natives of New Britain; melanotic sarcoma was not amongst these.

A good account of melanotic tumours in dark-skinned races is given by Smith and Elmes[3] in a record of 500 tumours in Nigerian natives. In this series melanotic sarcoma was encountered forty times, thirty of the growths having their site of origin in the foot. These writers refer to the wide histological variation seen in these tumours; all shapes of cell, from frank spindle-shaped to large flat polygonal cells, are seen. This is a feature which characterized the case here recorded.

Summary.

A case of melanotic sarcoma of the foot in a native of New Britain is recorded. Recurrence after local excision was first noticed about one year later, and the patient was in good general condition without evidence of metastases when last seen, about fifteen months after the initial operation. Reference is made to one unpublished case of melanoma in a native of Admiralty Island.

Acknowledgement.

I have to thank Dr. E. T. Brennan, Director of Public Health, for permission to publish this communication.

References.

(1) C. M. Deland, L. B. Bull and J. B. Cleland: "Epithelioma of Rodent Type in a Native of Bougainville", THE MEDICAL JOURNAL OF AUSTRALIA, June 10, 1933, page 712.

(2) G. R. Penington: "Observations on Tumours in New Britain Natives", THE MEDICAL JOURNAL OF AUSTRALIA, September 16, 1933, page 374.

(3) E. C. Smith and B. G. T. Elmes: "Malignant Disease in Natives of Nigeria: An Analysis of Five Hundred Tumours", *Annals of Tropical Medicine and Parasitology*, Volume XXVII, Number 4, 1934, page 461.

Reviews.

THE SHEEP'S BRAIN.

BRIGGS'S "Anatomy of the Sheep's Brain" fulfils well the purpose of the author to provide a dissection guide and short description of the gross anatomy of the brain for students entering upon a junior course of practical zoology.[1] The choice of the sheep's brain is a happy one for the purpose, material being readily accessible and quite adequate for general anatomical study. The description of the macroscopic structures is concise but clear, and the illustrations are good. It does, however, seem a pity that in one text figure letters are used to indicate parts, whereas in the others and in the plates the names are conveniently written in full. The arrangement of the plates at the end of the book does not add to the ease of reference while the text is being read.

The absence of adequate indications of the connexions of the brain, even without any indication of their physiological role, is to be regretted, especially for use of the book by students in veterinary science, for whom it is otherwise written in sufficient detail. For them the sheep's brain is quite a suitable type. The comparisons occasionally given with the brain of man appear to be unnecessary and not even helpful.

Both the author and the publishers are to be congratulated on the production of a very useful book at quite a moderate price.

Notes on Books, Current Journals and New Appliances.

A HAND-BOOK ON SEX HORMONES.

IN the issue of August 19, 1939, we published a note on "Ciba Handbook Number IV" issued by the Society of Chemical Industry in Basle. This book, which deals with the sex hormones, is issued to the medical profession only. A second edition has now been published[2] and includes some important additions to the first. This edition consists of five parts—the first four, as in the first edition, being concerned with historical matters, chemical and biological considerations, special actions of clinical significance and treatment; the fifth, a new part, is an appendix dealing with the percutaneous application of androgens and œstrogens and the oral administration of luteal hormone. While the proprietors point out that they are sceptical about many of the results that are claimed for treatment with sex hormones, they regard results reported from reliable sources in the treatment of dysmenorrhœa and sterility in the male (conditions not dealt with in the first edition) as warranting their inclusion. Forty-three new references have been added to the bibliography, the total being now over two hundred. Medical practitioners may obtain a copy of the hand-book by making written application to the society's representative in Australia, Box 271 DD, G.P.O., Sydney.

[1] "Anatomy of the Sheep's Brain: A Laboratory Atlas for Students of Zoology", by E. A. Briggs, D.Sc.; 1939. Australia: Angus and Robertson Limited. Demy 8vo, pp. 49, with eight plates and four text figures. Price: 6s. net.

[2] "The Sex Hormones: A Survey of their Chemical Constitution, Biological Properties and Clinical Applications, with a Classified Scheme of Dosage"; Second Edition; 1940. Basle: Society of Chemical Industry; Australia: J. M. Harkness (Sydney). Demy 8vo, pp. 128, with illustrations.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 14, 1940.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE TUBERCULOSIS PROBLEM IN AUSTRALIA.

INTEREST in the problem of tuberculosis in Australia received a great fillip at the fifth session of the Australasian Medical Congress (British Medical Association) held at Adelaide in 1937. Dr. M. J. Holmes, of the Commonwealth Department of Health, provided the greatest stimulus by his address at the plenary session. He showed that Australians had no reason for any feeling of satisfaction or of pride in having a death rate from tuberculosis that was lower than the rate of most other countries. The decline of the mortality rate from tuberculosis in Australia was no more pronounced, he stated, than in Scotland, a country with little of the natural advantages in climate and general living conditions that Australia possessed. He described our preventive measures as incomplete, ill-balanced and piecemeal. Dr. Holmes's contribution, which was published in this journal in the issue of November 6, 1937, is just as stimulating today as it was when it was published. There is no need to restate his general conclusions or to traverse his arguments; but we would recall the challenge in his declaration that in the past many diseases have been completely wiped out with knowledge less extensive than we possess today with regard to tuberculosis. Since 1937 investigators have not been idle, and most of the activities that were predicted in these pages for the National Health and Medical Research Council have taken place.

At a recent meeting the Council had before it a review by Dr. R. E. Richards, of the Commonwealth Department of Health, of the research activities into tuberculosis in Australia towards which the Council had made grants in 1938 and 1939. One of the investigations considered was that made by Dr. Douglas Anderson, an account of which was given in a series of "studies" published in this journal in June of this year. This survey, necessitating as it did the testing of 7,000 persons, was one of the most extensive of which we have knowledge, and since it was from start to finish the work of one man, its economical nature should be emphasized. Of the 7,000 persons tested by Dr. Anderson, 5,822 were regarded as representing a fair cross-section of the population; and it is important to note that the average age at which primary tuberculous infection occurs "is substantially later in Sydney than in other parts of the world where surveys of the general population have been made, and has become perceptibly later in the last fifteen years". It must also be pointed out that Dr. Anderson's survey was directed at the discovery of infection and not at the revelation of manifest disease. This does not imply that the survey did not yield information of the greatest value. For example, we learn that at least one-fifth of the urban population remains uninfected by the tubercle bacillus at the age of forty; and also that nearly one-half of the home contacts are infected by the age of five years and nearly two-thirds before they reach the age of puberty. In this connexion we note that Dr. Douglas Galbraith, in a report to the National Health and Medical Research Council on the problem of tuberculosis in the United States of America, states that in that country the incidence of tuberculous infection in children under five years of age is five times as great among contacts as among non-contacts. Opie found at the Phipps Institute in 1935 that 80% of the children in homes where tuberculosis was present were infected in the first five years of life. Unfortunately, Dr. Anderson's survey missed by about a year the possibility of its being carried out in conjunction with a miniature radiological survey. (For a discussion on this method of examination readers are referred to the leading article in the issue of this journal for March 30, 1940.) If all persons who had reacted to the Mantoux test had been subjected to an X-ray examination the survey would have been of more value from both the public health and the epidemiological points of view. Incidentally it would have provided a much more worth-while examination for the large number of people who volunteered to act as subjects, though it was clearly explained that the test would be of little or no personal value to them.

The National Health and Medical Research Council also had a report from Dr. H. W. Wunderly of a case-finding survey made by him among young women of the fifteen to thirty age group. Dr. Wunderly's report is published in this issue. It will be remembered that it was among women of the fifteen to thirty age group that the survey made by Dr. M. J. Holmes at the Adelaide congress revealed such a large number of infections. Mantoux skin tests were performed by Dr. Wunderly on 3,056 women of the fifteen to thirty age group; of these, 1,110 (35%) reacted and 1,946 (65%) did not react. The 1,110 who reacted were submitted to X-ray examination and 61 cases of active or probably active pulmonary tuberculosis were revealed. Particular stress should be laid on Dr. Wunderly's statement that only in exceptional cases was it claimed that a lesion could be classed as active on one X-ray examination. Readers will note that the presence of a cavity was accepted as a sign of activity, but that in other cases the findings on the examination of a series of photographs, the clinical history, the findings on clinical examination and the results of laboratory investigation were all taken into account. The immediate result of the survey is shown in the fact that 61 young women whose

condition was diagnosed as active tuberculosis, and who were in the early stage of the disease, were all "saved from developing a disabling pulmonary tuberculosis". Two sentences from Dr. Wunderly's report form a surprising contrast. We read: "There was the fullest cooperation on the part of the young women and of their employers." And in contrast: "One or two radiologists felt that the X-ray work should not have been done by a physician, and although several of the cases were later photographed by them they did not wish to see the earlier photographs." Dr. Wunderly has tried to answer two questions: first, why the incidence of pulmonary tuberculosis is so high among young women of the age group fifteen to thirty years; secondly, why the incidence in Adelaide is high when compared with other cities of the Commonwealth. The answers given by him to these questions are arresting. Whether they offer a complete explanation may be debatable; but it is at least certain that stricter measures should be taken to protect the general public from contact with persons suffering from "open" tuberculosis, and that the standard accepted as indicating an arrest of disease should be raised.

Two other surveys were brought to the notice of the Council by Dr. Richards. The first was a report from Dr. Eric Cooper on the incidence of pulmonary tuberculosis among university students and hospital nurses at Melbourne; this investigation is still proceeding. The second was an investigation carried out by the Department of Public Health of New South Wales into the condition of 45 bulk wheat handlers. Dr. Richards also pointed out in his review that it is estimated that three-quarters of the population of New South Wales have been at some period of life infected with tuberculosis and have successfully resisted the infection. It is not in childhood in Australia that the risk of infection is greatest, but in adult life, and greater precautionary measures must be taken to isolate persons suffering from open tuberculosis. Identification Dr. Richards describes as a comparatively simple matter if use is made of skin tests, X-ray examinations and cultivation of the tubercle bacillus by the method on which Dr. Reginald Webster has made a report to the Council.

It is clear that since Dr. Holmes made his indictment in 1937 of our measures to prevent tuberculosis, an attempt has been made to gauge the extent of the problem. It is also clear that infections must be discovered in their preclinical stages and infected persons must be prevented from developing manifest disease. As one observer has put it, we are devoting our energy to the treatment of disease at the wrong end. Only by means of surveys will it be possible to detect tuberculosis in the preclinical stage. The National Health and Medical Research Council evidently recognizes this, for it adopted as one of its resolutions a statement set out in one of the appendices "as outlining the principles upon which the campaign against tuberculosis should be based". Further: "These recommendations comprise, in the considered opinion of the Council, the measures necessary for the control of tuberculosis." The appendix in question is summarized in the following terms:

1. The work of Tuberculosis Clinics in relation to the homes in which tuberculosis exists should be supplemented by wider employment of the Mantoux test, X-ray examination and bacteriological diagnosis and control by direct cultivation.

2. The control measures in the general community should include: (*a*) mass surveys in certain age-groups (0-5 years and 15-30 years). (*b*) Special surveys in certain industries and occupations. These surveys should employ: (i) Routine Mantoux testing. (ii) Routine X-ray examination of positive reactors, using miniature radiography. (iii) Bacteriological diagnosis and control by direct cultivation.

3. A large increase in accommodation will be necessary.

4. A system has been outlined comprising chest hospital plus Tuberculosis Clinic working in conjunction with sanatoria, preventoria and industrial colony.

5. Careful attention is necessary to aspects of nutrition and environment of patient and contacts, and suitable provision should be made for this.

6. Special precautions should be taken to prevent contraction of disease under special hazards. Suggested precautions in relation to nursing trainees are set out.

Having adopted the statement summarized in this way, the Council continues with its resolution:

> Two serious difficulties present themselves—(*a*) Money is necessary for the accommodation which is essential for patients and contacts and for associated measures. (*b*) The economic condition of infected families must be adjusted.
>
> It is not possible for these difficulties to be entirely met by any co-ordination of existing voluntary agencies. Unless these two difficulties are removed the measures outlined cannot be brought fully into effect and the success of any campaign against tuberculosis must be correspondingly handicapped.

No one has ever doubted that the elimination of tuberculosis will cost money; nor will anyone doubt that the money can be found for this purpose if those in control of money really want to find it. The State is spending enormous sums in pensions and in hospitals for the tuberculous; when tuberculous infection becomes less, less will spent. It was shown at the Adelaide congress that if tuberculous infection at the present time was as widespread as in 1880, and if the same measures were adopted as are adopted at present, the amount of money spent by the State would be four times as great as it is today. Money spent on the prevention of tuberculosis today is an insurance premium for health in the future. On the evidence it would be justifiable to conclude that if no more than the present rate of expenditure is maintained a further improvement may be expected. This would be an ignoble attitude, for we know more than we did—the recent surveys have taught us something—and we may be quite certain that if we do not try to progress we shall go backwards. It has even been suggested that if complete provision cannot be made for persons discovered by surveys to be in the preclinical stage of the disease, no more surveys should be made. This view is untenable. Surveys reveal persons with "open" infections as well as those in the preclinical stage. The undiagnosed "open" infection is much more dangerous than the one already diagnosed; and if persons in the preclinical stage are discovered, they can, even with existing facilities, be supervised in many cases and prevented from advancing in the disease and from becoming infective. Again, the surveys have shown that nurses and medical students who do not react to the Mantoux test are in grave danger, and the same is true of other non-reactors who are compelled to remain in contact with persons suffering from "open" infections. Surveys must therefore be continued both in the general community and among those exposed to more than the ordinary hazards of infection. The National Health and Medical Research Council has stated as much as this, but it gives no indication in the

present report of its immediate intentions in this regard. From the report of the recent meeting we are justified in expecting a continuance of surveys, both general and special, and also some attempt to put into effect the Council's other recommendations. The Minister for Health, the Honourable H. V. C. Thorby, assured the Council in his opening address to members that he was prepared to cooperate with the Council in every possible way to do the utmost to eradicate tuberculosis. We believe that he meant what he said. With his help the National Health and Medical Research Council can probably do more than any other organization towards the attainment of our ideal—the eradication of tuberculosis from Australia.

Current Comment.

THE GERMAN MEASLES AND THE RECENTLY PREVALENT SORE THROAT.

In this country recently, as in the Old World a few months ago, at least four unpleasant minor maladies have been epidemic and have caused great inconvenience to many people, both directly and indirectly. These maladies have been a kind of head cold of unusual virulence, a milder kind of cold followed by a cough which lasted for a long time afterwards, a third kind of "cold" characterized by really extremely sore throat of several days' duration, and finally the German measles. There is reason to think that both the laity and the profession have been inclined, even in military camps, to treat these diseases with much less respect than has been accorded to them in days gone by, particularly as regards the time to be spent in bed by the sufferer, and, in the case of the German measles, the quarantine period. At the same time every doctor must have seen occasional patients who have been quite ill with the German measles. On both sides of the world complications of this disease have been frequently met with which do not appear to have been anything but rare for very many years; these complications are principally pains in the limbs, which may appear to be neuritic, and acute non-suppurative arthritis, flitting from joint to joint.

Some interesting observations have been made in England on the German measles and on the sore throat disease, recorded chiefly in letters to the medical journals; some of these, though made independently, fit in with one another remarkably like the pieces of a jig-saw puzzle. G. M. W. Hodges, writing in *The British Medical Journal* of March 30, 1940, appears to have been the first to report the occurrence of brachial neuritis after rubella, while C. Suffern reported polyarthritis as a complication in *The Lancet* of the same date. Similar reports were soon forthcoming. A. French suggested in *The British Medical Journal* of May 11 that the "neuritis" was "actually acute or subacute rheumatism associated with a streptococcal erythematous rash misdiagnosed as the ubiquitous rubella". Hodges replied (convincingly) the following week, insisting on his original diagnosis; but he added that he had heard that "in the Cambridge Pathological Laboratory a streptococcus has been grown in large numbers from every case of rubella examined". The following week again W. W. Wildman and W. B. Teasey, naval surgeons, reported epidemics coincident in time of German measles and of an acute cold with sore throat in one of His Majesty's ships at sea. No rating who had the "acute cold" subsequently developed rubella, and *vice versa*. This suggested to R. P. Mattock, writing in the same journal on June 1, that the German measles and the sore throat disease might be closely related. Certainly in this country, of late, arthritis and pains in the limbs have been complications common to both maladies.

We are not aware that any comparative hæmatological or bacteriological studies of the recent epidemics of rubella and sore throat have so far been published, although it has been stated in newspapers that the sore throat disease is at present under investigation at the Institute of Medical and Veterinary Research at Adelaide. It will be strange indeed if these diseases have swept across the world without any relationship that may exist between them having been exactly defined. Still, not only in yielding up the secrets of their pathology, but in their therapeutics these have been refractory diseases, and the art of the nurse has brought more comfort to many sufferers than the science of the physician. However, newly introduced analgesic demulcent tablets have been afforded a good opportunity to prove their value for very sore throats. For sore throat also aspirin is said to have been much more effective than sodium salicylate, while for pains in the limbs and joints the well-tried phenazone and its analogues have been recommended. Fortunately the most uncomfortable complications have not been lasting, and at their height the physician has felt confidence in prognosis and treatment in view of similarities of the symptoms to those which much more commonly complicate other infectious diseases.

PROTHROMBIN CONTENT OF PRESERVED BLOOD.

Blood transfusion has been so simplified and its therapeutic field has been so extended that it has become a commonplace procedure. Blood must be as readily available for transfusion as, say, saline solution. It is not yet possible to buy a pint of blood over a pharmacy counter; but one day it may be. The storage of blood is more and more widely practised. There is no need to repeat here the various changes that have been found to take place in stored blood nor the precautions that have to be taken; we are concerned for the moment with the changes in the prothrombin content. According to Rhoads and Panzer, the prothrombin content falls rapidly and is so slight after a week as to be practically useless to a person suffering from prothrombin lack. John Reinhold, Eleanor H. Valentine and L. Kraeer Ferguson disagree with this view and back their opinion with the results of their own experimental work and that of De Gowin, Harris and Plass.[1] They examined 17 specimens of preserved blood at intervals over a period of three to five and a half weeks. On an average the prothrombin content diminished to 73% of its original value within three days and to 55% in six or seven days. Twelve of the specimens contained less than 50% of their original prothrombin content at 13 to 17 days; but two retained their full value up to this time. In seven specimens there was little or no hæmolysis after periods considerably longer than seventeen days, "and it may be significant that in these, suitable in appearance for transfusion, the prothrombin concentration remained at an average of 52, 48 and 53% respectively at 3, 4 and 5 weeks". However, it should be noted that after six to eight days only, the prothrombin content of one specimen had fallen to 32% and of another to 34% of the original value. Reinhold, Valentine and Ferguson calculate that if 500 cubic centimetres of blood stored for three days were given to a patient with a blood volume of 7,000 cubic centimetres and a prothrombin value of 15%, the patient's prothrombin value would be increased to 19%; whereas a transfusion with the same amount of fresh blood would raise it to no more than 20·6%. At the hospital where these experiments were conducted, 90% of preserved blood is used within two or three days; during this time the diminution in the prothrombin content is negligible. The clinical experience of the results of transfusion of stored blood is in accord with experimental evidence. The next steps in the elucidation of this interesting problem will be: (i) the devising of a simple means of ascertaining readily the prothrombin content of a sample of blood, and (ii) a determination whether it is the prothrombin content only that is responsible for the total increase in the prothrombin content of the patient's blood or whether there is some other factor in the transfusion.

[1] *The American Journal of the Medical Sciences*, June, 1940.

Abstracts from Medical Literature.

PHYSIOLOGY.

The Metabolism of Alcohol.

M. GRACE EGGLETON (*The Journal of Physiology*, May, 1940) describes the results of work done on some of the factors which may affect the metabolism of alcohol in the body of anæsthetized cats. This paper is preceded by another which deals with the technique of the methods of analysis employed, together with an examination of the times necessary for equilibration. Comparison of results obtained on animals under light "Nembutal" anæsthesia with those obtained by other workers on unanæsthetized animals shows that this anæsthetic has little, if any, inhibiting action on the metabolic rate of alcohol. The rate in cats was found to be directly dependent on the concentration of the alcohol in the body, increasing about 30% for every 100 milligrammes per 100 millilitres of increase in plasma alcohol concentration. This relationship was demonstrated only when the metabolic rates were expressed in terms of liver weight rather than of body weight or body surface. The metabolic rate of alcohol per unit of liver weight in animals previously fed with alcohol was significantly lower than in normal animals. The metabolic rate of alcohol was increased by the addition of alanine to the body. The author suggests the simplest explanation, that of delayed absorption and increased rate of metabolism of the alcohol due to the specific dynamic action of certain foodstuffs when alcohol is taken with food, rather than that of Widmark, who suggests combination of the alcohol with some of the food elements.

Injury to Ileal Mucosa by Distilled Water.

CLARENCE DENNIS (*The American Journal of Physiology*, April, 1940) describes the injury to the mucosa of the lower portion of the ileum occasioned by contact with distilled water. Interference with the ability of the epithelium to do osmotic work and with its impermeability to sulphate has been demonstrated. Microscopic evidence of injury to the entire jejunum and ileum is also tendered. Certain published studies concerning the activity of the gut, which need reconsideration, have been mentioned. Practical application of the findings discussed in this paper is found in criticism of the use in clinical practice of distilled water for irrigation of the small intestine. It is suggested that some isotonic solution, such as 0·9% sodium chloride solution, be used instead of distilled water for irrigation of the small intestine in bowel obstruction.

Diet and the Insulin Content of the Pancreas.

C. H. BEST, R. E. HAIST AND JESSIE H. RIDOUT (*The Journal of Physiology*, November, 1939), working with Wistar rats, describe experiments carried out to determine the effect of various diets on the insulin content of the pancreas. Methods used in the assay of the pancreatic material are described. The results after anæsthesia with urethane or "Sodium amytal" were compared with those obtained after stunning, and although the sugar content of the blood was definitely higher in the group which received urethane, no significant difference in the insulin content of the pancreas could be noticed between the three methods. Fasting or the feeding of diets rich in fat was shown to produce a definite decrease in the insulin content of the pancreas. While an abnormally low calorific intake may be one of the factors producing this result in the animals given the diet rich in fat, it is not the only one, since those receiving a certain calorific intake as fat show a much greater decrease in insulin content than those provided with the same calorific intake in the form of carbohydrate. The insulin content of the pancreas, depleted by fasting, is restored to normal within six days by the feeding of a well-balanced diet. Carbohydrate alone effects a partial restoration; but fat alone produces no rise in insulin content. Vitamin B_1 deficiency does not affect the insulin content of the pancreas in any specific manner. The possible relationship between the low insulin content of the pancreas and the altered carbohydrate metabolism of animals which have fasted or have been fed diets rich in fat, is discussed.

Penetration of Cervical Mucus by Spermatozoa.

J. K. LAMAR, L. B. SHETTLES AND E. DELFS (*The American Journal of Physiology*, May, 1940) have published a description of the results obtained by means of a new method for the examination of human cervical mucus with regard to its penetrability by spermatozoa. Fifty-eight subjects were examined and the results confirmed the findings of Seguy and Vimeux. There is a cyclic production of a glairy translucent cervical mucus at about mid-cycle, which renders the cervix temporarily penetrable by spermatozoa. The limits of the occurrence of this phase were between the ninth and the nineteenth days, although the actual period of penetrability may be restricted to about four days. The menstrual discharge was penetrable by spermatozoa, but the mucus during the rest of the cycle was impenetrable. Correlated with the mid-cycle peak of penetrability are the peak of freedom from leucocytes, the peak of pH (except during the menstrual flow), the peak in the amount of mucus secreted, and the height of conditions favouring greatest longevity of spermatozoa in the mucus. An investigation of the cervical mucus over the cycle, by the *in vitro* method, is suggested in cases of otherwise unexplained sterility. A field of usefulness for artificial insemination may be developed in sterility due to a persistent impenetrable cervical mucus.

Composition of Pericardial and Peritoneal Fluids.

F. W. MAURER, M. F. WARREN AND C. K. DRINKER (*The American Journal of Physiology*, June, 1940) report analyses of the pericardial and peritoneal fluid of dogs, monkeys, cats, hens, ducks, rabbits and humans, the last material being obtained from autopsies. They draw attention to the lack of any literature on the subject; they have demonstrated the existence of reasonable quantities of pericardial fluid in even a small animal such as a rat. These fluids are relatively rich in protein, the amount being also constant. In the human autopsies the percentage of protein obtained varied from 0·78% to 6·68%; the fluids were regarded as abnormal both in protein content and in volume. The autopsy findings are given. The ratios of albumin to globulin were determined in the pericardial and peritoneal fluid from some of the dogs and rabbits, but little or no similarity was found to the ratio determined in the serum of the same animals. The presence of fibrin was shown in both pericardial and peritoneal fluids by the rapid clotting of the fluids immediately after removal from the animal. The chloride ratios of the fluids and the serum of the animal compared closely with the theoretical ratios calculated on the assumption of a Donnan equilibrium. It was shown that the protein contained in these extravascular fluids may easily be derived from the protein of the blood stream by simple diffusion across the blood vascular membrane.

Gastric Carbonic Anhydrase.

FOLLOWING on the work of Davenport and Fisher, who discovered large amounts of carbonic anhydrase in the gastric mucosa of animals during their study of the secretion of hydrochloric acid, H. W. Davenport (*The Journal of Physiology*, November, 1939) reports the results of a more detailed study of the distribution of the enzyme in the stomach wall. Estimations of carbonic anhydrase were made on slices of samples stamped from the gastric mucosa by a cork borer; by the counting of the parietal cells in both adjacent slices the distribution of the enzyme in the stomach wall was indicated and its association with the parietal cells was proved. The enzyme is present in lesser amounts in the pyloric and cardiac portions of the stomach wall; it thus follows the distribution of the parietal cells. The concentration of the enzyme in the parietal cell may be five times higher than in blood. It is present in the surface cells of the mucosa in about one-tenth of the concentration in the parietal cells. References are given to the results of numerous workers who have demonstrated some connexion between carbon dioxide and gastric secretion. In the light of the great concentration of carbonic anhydrase now shown to be present in the gastric mucosa, these findings take on added interest.

Duration of Insulin Action.

PAUL O. GREENLEY (*The American Journal of Physiology*, April, 1940), working on depancreatized dogs, has made a study of the rate of destruction of insulin in the body. The results would indicate that for these animals the duration of the action is proportional to the logarithm of the size of the dose. The rate at which insulin is being destroyed in the body at any instant is proportional to the amount present in the body at the time. The average rate of destruction is four-tenths of this amount per hour, or one nine-thousandth of the amount per second.

Effects of Anæsthesia on the Blood Supply to the Hypothalamus.

A. E. LAIDLAW AND M. A. KENNARD (*The American Journal of Physiology*, June, 1940) discuss the effect of anæsthesia with ether and with barbiturates on the size of the capillaries of the hypothalamus. Monkeys were used as laboratory animals. After the animal

had been held in a state of surgical anæsthesia for a period of fifteen to sixty minutes, injection with India ink and gelatin was started according to Findley's adaptation of Pfeifer's method. After being hardened in formalin the brain was removed from the skull, sectioned and stained. Following barbiturate anæsthesia numerous dilated capillaries are seen in the supraoptic and paraventricular nuclei of the hypothalamus; but after ether relatively few capillaries are open, and these appear constricted. In contrast, the capillaries of the cortex are more dilated by ether than by the barbiturates. These findings are offered as further evidence for a hypothalamic site of barbiturate action. The authors draw attention to the recognized similarity of this action and deep sleep.

BIOLOGICAL CHEMISTRY.

Effect of Zinc on Phosphatases.

E. HOVE, C. A. ELVEHJEM AND E. B. HART (*The Journal of Biological Chemistry*, June, 1940) have reported metabolic studies designed to elucidate the role of zinc, which is an element essential to the life and growth of rats. In zinc-deficient rats the intestinal phosphatase content was considerably lower than in normal controls. This may be related to the previously established decrease in pancreatic tryptic activity during zinc deficiency. Crude intestinal phosphatase activity was increased 40% to 100% by the addition of zinc ions *in vitro*, while crude kidney and bone phosphatase activity was progressively inhibited by zinc. After dialysis all three enzymes were inhibited by zinc. Readdition of the dialysate from intestinal phosphatase preparations restored the ability of zinc to activate the dialysed enzyme. The dialysable zinc coactivator was a product of mucosal tissue autolysis. The same coactivating property was demonstrated by all the pure α-amino acids tested. It is considered that the *in vitro* relations between zinc, amino-acids and organic phosphoric esters may explain the previous observation that amino-acid absorption from the intestine is delayed in zinc-deficient rats.

Renal Function in Gout.

F. S. COOMBS, L. J. PECORA, E. THOROGOOD, W. Y. CONSOLAZIS AND J. H. TALBOT (*The Journal of Clinical Investigation*, May, 1940) have investigated kidney function in 22 cases of gout. The methods of studying renal function were: determination of the non-protein nitrogen content of the serum; testing of the ability to concentrate solids; measurement of the excretion of phenolsulphonephthalein dye; pyelographic examination after the intravenous injection of "Neo-iopax"; and estimation of the rate of clearance of insulin, creatinine, urate, sodium and chloride. Most of the gouty patients showed some evidence of renal damage. The earliest change was inability to concentrate solids. In the absence of severe renal impairment, all except 10% of the urates which were filtered through the glomeruli, were reabsorbed by the tubules. When severe renal impairment was present, reabsorption of urates by the tubules was depressed and clearance tended to be maintained. No constitutional inferiority of the kidneys to excrete urate was demonstrated. Cinchophen and "Salyrgan" caused a diminution in tubular reabsorption of urate and an increase in urate clearance. Colchicine did not appear to influence the renal elimination of urates. The authors believe that the kidney changes in patients suffering from gout are the result and not the cause of the metabolic dyscrasia.

Inhibition of Growth by Substances in the Liver.

A. M. BRUES, Y. SUBBAROW, E. B. JACKSON AND J. C. AUB (*The Journal of Experimental Medicine*, April, 1940) have described experiments with certain tissue constituents, extracted from liver, which are inhibitory to cell growth. The inhibitory material is probably adsorbed to colloids in the native state and can be freed from them by alcoholic extraction. One inhibitor was isolated and identified as ethanolamine, but this substance represented only a small proportion of the total inhibitory activity of liver extracts. The inhibitors have the following common properties, which suggest that they are of physiological significance in the regulation of growth: action over a wide range of concentrations at which other cell functions are undamaged; reversibility of action; presence in adult liver in concentrations near those which inhibit growth *in vitro*, while in embryo liver they are found only in much lower concentrations.

Creatine Formation.

H. BORSOOK AND J. W. DUBNOFF (*The Journal of Biological Chemistry*, February, 1940) have shown that, *in vitro*, liver slices of the cat, rabbit and rat are able to convert glycocyamine to creatine. In the experimental conditions the increase was five to twenty times the amount of creatine originally present in the slices. When methionine also was added the amount of creatine formed was increased by 50%. Methionine was the only substance among many tested which had this effect. In a later communication (*The Journal of Biological Chemistry*, July, 1940) the same authors have shown that the livers of the cat, dog, guinea-pig, frog, pigeon, rabbit and rat all possess the property of converting glycocyamine to creatine. The stimulating effect of methionine was found in some but not all animals. The kidney appeared to be unimportant (except in the pigeon) for the methylation of glycocyamine with or without methionine. These findings were interpreted as indicating that creatine normally is formed by the methylation of glycocyamine in the liver, although the possibility of other mechanisms and other sites of creatine formation is not excluded.

Alcohol in the Blood and Urine of Drunken Drivers.

DONALD F. BAVIS (*The Journal of Laboratory and Clinical Medicine*, May, 1940) has estimated the alcohol content of the blood or urine, or both, of 145 persons accused of drunkenness while driving. Specimens of blood and urine were examined simultaneously in 66 instances, specimens of urine only in 54, and specimens of blood only in 25. Definite correlation was observed between the alcohol concentrations in blood and urine, and it was found that for most drunken drivers the values for the urine fell between 0·2% and 0·3%, and for the blood between 0·15% and 0·25%. In 120 subjects good correlation was observed between the concentration of alcohol in the urine and the degree of intoxication noted clinically. Of those persons with a urine alcohol content between 0·2% and 0·3%, 88% were moderately intoxicated. The author considers that occasional discrepancies between alcohol concentrations and behaviour do not invalidate the significance of the results of the tests in police work.

Thiamine in Citric Acid Metabolism.

H. A. SOBER, M. A. LIPTON AND C. A. ELVEHJEM (*The Journal of Biological Chemistry*, July, 1940) have studied the relation of thiamine (vitamin B_1) to citric acid metabolism. Rats receiving a thiamine-deficient diet showed a decreased excretion of citric acid during acute deficiency and a decrease in the ability to transform injected succinic acid into citric acid. Within four to six days after the administration of thiamine to rats receiving a diet deficient in this substance there was a tenfold rise in citric acid excretion. The level then dropped slowly and normal values were restored in ten to fourteen days after the initiation of therapy. It was concluded that thiamine pyrophosphate (cocarboxylase) is essential for the synthesis of endogenous citric acid from its precursors, and may possibly also play a role in the catabolism of citrate.

Metabolism of Iron.

M. E. AUSTONI AND D. M. GREENBERG (*The Journal of Biological Chemistry*, June, 1940) have studied, with the aid of radio-active iron as an indicator, the absorption, distribution and excretion of iron in normal and in iron-deficient rats. It required about twelve hours for a single dose of iron to pass from the stomach and small intestine; passage through the gastro-intestinal tract was significantly slower in the anæmic rats. The anæmic animals eliminated less of the administered iron in both fæces and urine. During a ten-day period the normal animals retained about 30% of the administered iron, while the anæmic animals retained 50%. The greater part of the elimination through the fæces and urine occurred within forty-eight hours. The specific accumulation of the absorbed iron per gramme of tissue was greatest in the bone marrow, blood, spleen, liver and heart. The total accumulation was greatest in muscle and blood. After ten days the radio-active iron had nearly disappeared from the muscle and blood of the normal rats, but considerable accumulation had occurred in the muscle (25% of the administered dose) and blood (14% of the administered dose) of anæmic animals.

P. F. HAHN, J. F. ROSS, W. F. BALE AND G. H. WHIPPLE (*The Journal of Experimental Medicine*, June, 1940) have observed that the formation of hæmoglobin in the anæmic dog takes place at a very rapid rate. Radio-active iron is detectable in the circulating red cells four hours after feeding. The absorbed radio-active iron is entirely converted into hæmoglobin within four to seven days under standard anæmic conditions. When erythrocyte production has been accelerated by the giving of iron or by diet factors, or when the dose of iron is very small, the dog can utilize for hæmoglobin formation almost all the absorbed radio-active iron in two to three days. In general, absorption of radio-active iron in anæmic dogs is more efficient in small doses in single feeding experiments. As the iron intake is increased, the percentage absorption rapidly falls.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

XI.

CONFUSIONAL INSANITY.

This is a strange repose, to be asleep
With eyes wide open, standing, speaking, moving,
And yet so fast asleep.

CONFUSIONAL INSANITY is a state of mind rather than a definite form of mental disease. It varies from the condition in which normal people find themselves when they have difficulty in concentrating their attention when tired, to complete confusional stupor when the patient is entirely oblivious of his surroundings. Children who become delirious when their temperature rises are suffering from confusional insanity.

Causes.

This form of insanity is due to some interference with the mechanical action of the brain. The relation between brain and mind has been debated since the days of Aristotle; but it is generally agreed that interference with the brain as a machine may cause interference with the action of the mind. I lay stress upon the word "may", as cerebral tumour, cerebral embolism and even severe cerebral trauma may occur with no impairment of the mental faculties.

The symptoms arising in confusional insanity can be readily understood if it is realized: that the mind no longer appreciates the information conveyed by the organs of special sense; that the patient's knowledge of time is distorted, the past and present becoming intermingled; that the real and imaginary are indistinguishable and the patient alternates between states of dreaming, terror and lucidity.

The chief pathological causes of this mental state are:

1. Gross brain lesions, cerebral embolism or hæmorrhage, cerebral tumours, arteriosclerosis, cerebral concussion or traumatism, cerebral syphilis and epilepsy.

2. Pollution of the cerebral blood supply in septicæmia, including puerperal sepsis, pneumonia, influenza, diabetes and renal conditions.

3. Depletion of the cerebral blood supply in arteriosclerosis, cardiac failure, anæmia. This condition is very common in senile patients a few days before death.

4. External poisons, such as alcohol, lead, belladonna, carbon monoxide and probably other gases.

5. Physical exhaustion due to loss of sleep and insufficient food.

6. Emotional shock. This is a very rare cause and usually is accompanied by one or more of the above-mentioned pathological conditions.

Symptoms.

The patient may show gradually increasing confusion of thought, becoming worse over weeks or months, or the attack may come on with extreme rapidity, the patient being in a state of acute delirium in a few hours.

He is restless, tries to get out of bed and will wander aimlessly into the street in his nightclothes. He does not recognize his relatives nor his surroundings, he talks incoherently and sometimes gazes into space with wide open, vacant eyes. When addressed, he may take no notice or may shrink away. He may not obey simple commands like: "Give me your right hand"; but frequently when the command is repeated two or three times he obeys in a hesitating and bewildered manner.

He may refuse his food and resist when anyone tries to feed him; sometimes he will swallow when food is placed in his mouth.

His actions indicate that he hears voices talking to him and sees visions; but usually he is unable to give information about them. He frequently evinces apprehension or terror, and may attack people coming into his room, as he is unable to distinguish between hallucinations and reality.

He is quite unable to give information about himself, does not recognize his surroundings, and his sense of time is lost.

The patient sleeps badly, and the restlessness usually becomes worse at night, owing to the increasing cerebral anæmia and the lack of external stimuli giving free play to the visual and auditory hallucinations.

It is this form of mental disease that shows the "lucid intervals", so beloved of lawyers, and occasionally for an hour or two at a time the patient becomes clear and rational.

Physically the patient looks ill; he has a strained bewildered expression, rapidly loses weight, the pulse becomes rapid and feeble, the mouth and tongue become dry and brown, and there is often a slight rise of temperature apart from cases due to some febrile disease.

Prognosis.

Prognosis depends on the cause of the condition. In an acute disease like pneumonia or septicæmia the fact that the patient is difficult to manage makes the prognosis graver; but if the patient's strength can be maintained and the physical condition remedied, the prognosis is good. As the physical condition improves, so will the mental confusion gradually pass away.

Treatment.

First a thorough physical examination is made, and this is sometimes extremely difficult on account of the non-cooperation or active resistance of the patient. The ophthalmoscope is the most valuable instrument in these cases and often the true condition cannot be suspected until papilloedema or albuminuric retinitis is disclosed.

If a physical disease is discovered this must be treated, and in addition the general principles of treatment may be summarized under five headings:

1. Get the bowels well open with suitable aperients.

2. Insist that the patient has plenty of fluid food, given by the stomach tube if necessary. This is particularly urgent in puerperal cases and on no account must the patient be allowed to go without food in the hope that she will be "better tomorrow". Peptonized milk, eggs, meat and fruit juices can be poured down the tube and must be given three or four times a day. Alcohol is most valuable in confusional insanity, and brandy should be given in all cases except those which present a clinical picture like that of a gross brain lesion or epilepsy. Fortunately tube feeding need not be continued for more than two or three days; after that the patient usually takes his food himself. Whether the food has to be given by the tube or the patient takes it himself, he must be given as much as possible. A well-trained nurse is very valuable in these cases, as she will be able to persuade a patient to take his food; if he has an incapable nurse he will have to be tube fed. An untrained nurse may report that the patient has taken a cupful of milk, when he has really taken a quarter of a cupful and the remainder has been scattered over the floor.

3. Of sedatives, potassium bromide, given in twenty-grain doses every two hours, is the most satisfactory, the interval between the doses being lengthened as the patient becomes quieter. Paraldehyde, two to four drachms, and other narcotics can be given at night in addition. In cases due to cerebral anæmia from cardiac failure, cardiac stimulants are much more effective than sedatives. Morphine is quite useless in confusional insanity, and hyoscine should be avoided unless the patient is so violent that he will damage himself or someone else. Sometimes a dose of one-fiftieth of a grain of hyoscine hydrobromide for a man or one one-hundredth of a grain for a woman is the only means by which a patient can be kept sufficiently quiet to enable him to be taken to hospital. Any dose less than this is useless.

4. In cases of confusional insanity I have found that injections of "Campolon", two cubic centimetres, given intramuscularly every second day, appear to be very beneficial. "Campolon" is very useful for patients who improve up to a certain stage but do not get quite well. I am now using it in all cases of confusion except those due to arteriosclerosis, gross brain lesions and senility.

5. When the patient has become convalescent, massage and ultra-violet rays are very useful.

After-Effects.

If the cerebral confusion is caused by an acute physical disease and the patient recovers from this disease, the results are most satisfactory. The patient gradually recovers his sanity, his periods of restlessness and confusion become shorter, and, except for a loss of memory for the period of confusion, he should make a complete recovery and there is no reason to anticipate a recurrence.

JOHN K. ADEY, O.B.E., M.B., F.R.A.C.P.,
Lecturer in Psychiatry, University of Melbourne; Medical Superintendent, Receiving House, Royal Park, Victoria.

Naval, Military and Air Force.

A CLINICAL MEETING IN PALESTINE.

A CLINICAL group has been formed in connexion with the Australian hospitals of the Australian Imperial Force serving in Palestine. The first clinical meeting of the group was held on July 13, 1940, at the First Australian General Hospital, Gaza Ridge. Forty-six medical officers were present, including: Colonel S. R. Burston, D.D.M.S.; Colonel H. C. Disher, A.D.M.S., Sixth Division; Colonel J. Steigrad, commanding the First Australian General Hospital; Colonel W. W. S. Johnston, commanding the Second Australian General Hospital; medical officers from the First and Second Field Ambulances, the First Casualty Clearing Station and other field units; officers of the Royal Army Medical Corps from the Sixty-Second British General Hospital, Jerusalem; Professor Adler, from the Hebrew University, Jerusalem, and Dr. Rachmilewicz, from the Hadassah Hospital, Jerusalem, who were present by invitation.

COLONEL S. R. BURSTON expressed to Colonel Steigrad and the officers of the First and Second Australian General Hospitals his appreciation of the holding of the meeting. He referred to the great value of such meetings to medical officers in the army, and said that particularly to those in field units and to those engaged in administrative duties the advantage of such opportunities for refreshing clinical knowledge could not be over-estimated. He hoped that more facilities would be available to enable officers of the Australian Army Medical Corps to keep in touch with medical and surgical work.

Hyperthyreoidism.

CAPTAIN R. H. MACDONALD showed a patient who had been receiving treatment for persistent ulcerative stomatitis; acute hyperthyreoidism had supervened while he was under observation. The patient was being prepared for operation. Captain MacDonald asked for suggestions with regard to the treatment of the inveterate ulceration of the mouth.

LIEUTENANT-COLONEL E. BRITTEN JONES agreed with the proposed operative treatment. He suggested that the stomatitis might resolve later, when the metabolic rate had been reduced.

LIEUTENANT-COLONEL A. S. WALKER showed a patient suffering from hyperthyreoidism, who had a peculiar coarse rolling tremor of the "intention" type. There had been no acute or recent increase in his hyperthyreoid state. Lieutenant-Colonel Walker said that the points at issue were the nature of the tremor, the question of treatment and the advisability of operation, which at the time did not appear to be indicated.

COLONEL W. W. S. JOHNSTON and MAJOR DEWAR, R.A.M.C., suggested a possible functional cause for the tremor. It was agreed that there was no indication for thyreoidectomy at the present time.

Jaundice with Erythronoclastic Anæmia.

MAJOR L. F. DODS showed a patient suffering from jaundice with erythronoclastic anæmia; his jaundice conformed well with the so-called acholuric type. The diagnosis was supported by a history suggesting biliary colic, by the presence of a rapidly decreasing anæmia, by evidence of splenic enlargement observed over a period of months, by some increase in the mean corpuscular volume, and by the presence of reticulocytosis.

LIEUTENANT HAMILTON, R.A.M.C., DR. RACHMILEWICZ, MAJOR DEWAR, R.A.M.C., and MAJOR I. M. MACKERRAS agreed that there was evidence of alterations in the architectural structure of the erythrocytes and of some slight increase in fragility. It was pointed out that there was no strong indication for splenectomy; but attacks of biliary colic might persist unless some action was taken.

Infective Hepatitis.

CAPTAIN E. L. DAVIS showed a patient suffering from infective hepatitis. The question of splenic enlargement was raised, and the findings were compared and contrasted with those of the preceding case.

LIEUTENANT-COLONEL E. BRITTEN JONES, PROFESSOR ADLER, COLONEL S. R. BURSTON, MAJOR DEWAR and DR. RACHMILEWICZ discussed the condition. The most important points touched on were: the epidemic and endemic nature of mild non-leptospiral jaundice in Palestine, the essential feature of the disease being a liver-spleen infection, the paucity of autopsy evidence, and the necessity for pursuing modern histological methods when autopsies were carried out in cases of jaundice, particularly with regard to examination for cell inclusions.

Hydronephrosis.

MAJOR T. Y. NELSON showed a patient suffering from hydronephrosis. He pointed out that although no ureteral stone was found, as had been expected when the ureter of the patient was being explored, a band had been discovered. Dr. Nelson hoped for the restoration of good renal function.

LIEUTENANT-COLONEL L. C. E. LINDON agreed that the prognosis should be good.

COLONEL S. R. BURSTON inquired as to the necessity of providing ureteral catheters in the urological equipment supplied to a general hospital. It was agreed that ureteral catheters were absolutely necessary.

Optic Atrophy with Sphenoidal Sinusitis.

CAPTAIN E. A. HEDBERG showed a patient suffering from optic atrophy with sphenoidal sinusitis. The patient had originally been admitted to hospital ostensibly almost blind, but with photophobia. This incompatibility had indicated a partly functional state in addition to a degree of optic atrophy, at least in one eye.

MAJOR DAVY described the operative treatment he had adopted. He said that he hoped for further improvement in the patient's condition.

MAJOR WALLIS, R.A.M.C., and LIEUTENANT-COLONEL LINDON also took part in the discussion.

Neurosyphilis.

CAPTAIN W. M. ADA showed a patient suffering from neurosyphilis. Signs suggesting cerebral involvement were present, causing loss of power in the left leg and also sensory changes in the leg. Some difficulty was being experienced in classifying the type of lesion, whether it was parenchymatous or meningo-vascular; but the paucity of findings in the cerebro-spinal fluid made the latter less likely.

MAJOR N. W. FRANCIS referred to other difficulties in diagnosis, particularly with regard to the comparatively short history available.

Exhibition of Microscopic Slides Illustrating Malaria.

MAJOR I. M. MACKERRAS showed a number of microscopic slides illustrating malaria.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 181, of August 29, 1940.

AUSTRALIAN MILITARY FORCES.

Army Headquarters.

Major (Honorary Lieutenant-Colonel) (Temporary Lieutenant-Colonel) C. H. Kellaway, M.C., Australian Army Medical Corps, from the appointment of Director of Hygiene, Army Headquarters, is appointed Director of Pathology, and to be Colonel (temporarily), 1st June, 1940. Captain (Honorary Major) M. J. Holmes, D.S.O., Australian Army Medical Corps, is appointed Director of Hygiene and to be Lieutenant-Colonel (temporarily), 1st June, 1940, *vice* Major (Honorary Lieutenant-Colonel) C. H. Kellaway, M.C., who relinquishes the appointment on 31st May, 1940.

NORTHERN COMMAND.

First Military District.

Australian Army Medical Corps.

To be Majors (temporarily)—Captains C. R. Morton and D. H. K. Lee, 24th July, 1940; Captains D. V. Sheil and A. F. Quayle, 26th July, 1940. *To be Captain (provisionally)*—John Lawrence Gorman, 16th July, 1940. Honorary Captains A. J. Kennedy, D. G. Picone and A. Fryberg are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally), 30th June, 1940.

Australian Army Medical Corps Reserve.

Reserve of Officers (A.A.M.C.).—To be Honorary Captains—Thomas George Hewitt, 1st July, 1940; Ian Stafford Ross Munro, 14th July, 1940; Cyril Cuthbert DeVaux Weedon, Stanley Holmes Martin and Alan Ernest Porter, 20th July, 1940, and Henry William Hackett, 1st August, 1940.

SOUTHERN COMMAND.

Third Military District.

Australian Army Medical Corps.

Captain (Honorary Major) M. J. Holmes, D.S.O., is appointed from the Reserve of Officers (A.A.M.C.), 1st June, 1940. The resignation of Captain (provisionally) H. C. Stone of his commission is accepted.

To be Captain (provisionally)—Osburn Bracewell Goyen, 8th July, 1940. Captain E. E. Price is appointed from the Reserve of Officers (A.A.M.C.), with regimental seniority next after Captain H. D. B. Miller, 1st July, 1940. Honorary Captain R. J. Farnbach is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally) with regimental seniority next after Captain (provisionally) V. Brand, 11th July, 1940. Captain C. E. Crooke is transferred to the Reserve of Officers (A.A.M.C.), 8th July, 1940.

To be Captains (provisionally)—George Vincent Rudd, 21st June, 1940; Donald Grant Duffy, 1st July, 1940; John Noel Brown and Victor Brand, 29th July, 1940. The undermentioned officers are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally) from the dates stated: Honorary Captain P. J. Parsons, 29th June, 1940; Honorary Captains P. G. Hayes, L. H. Ball and D. M. Brown, 30th June, 1940; Captain W. K. Davenport is transferred to the Reserve of Officers (A.A.M.C.), 29th July, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Clarence Leslie Halkyard, John Patrick Walsh, Gethyn Llewellyn Thomas, Ian Bruce Webster, Vivian John Taylor, Keith Henry Tweeddale, Leo Anthony Kennedy, Geoffrey Hilmorton Smith and Hartley Gibson, 21st June, 1940.

To be Honorary Captains—Philip Goodman, 8th July, 1940; Arthur Theodore Langley, Sydney John Newing and John Patrick Horan, 29th July, 1940.

Fourth Military District.

Australian Army Medical Corps.

To be Major (temporarily)—Captain E. F. Gartrell, 29th June, 1940.

To be Captain (provisionally) supernumerary to establishment pending absorption—Robert Stuart Wilkinson, 9th May, 1940. Major J. S. Verco is appointed from the Reserve of Officers (A.A.M.C.) and is supernumerary to establishment pending absorption, 10th July, 1940. Major F. L. Wall, M.C., is appointed from the Reserve of Officers (A.A.M.C.), 22nd July, 1940. Captains A. J. Blofeld-Moody and A. W. S. J. Welch are appointed from the Reserve of Officers (A.A.M.C.), 17th July, 1940, and 19th July, 1940, respectively. Captain C. H. Schafer is appointed from the Reserve of Officers (A.A.M.C.), 26th July, 1940. The undermentioned officers are appointed from the Reserve of Officers (A.A.M.C.), and to be Captains (provisionally) from the dates stated: Honorary Captains K. V. Smith, G. V. Smith, and M. R. Gold, 30th June, 1940, and R. H. Hamilton, 24th July, 1940. Captain R. N. Reilly is transferred to the Reserve of Officers (A.A.M.C.), 2nd July, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captains—Colin George King and Arnold Broughton Eckersley, 14th June, 1940; Arthur Henry Waterson and Gordon Baden Powell Loeser, 15th June, 1940; Harold Vernon Clarke, 17th June, 1940; Lancelot David Solomon, James Scollin and Wesley Melvyn Tiller Marshman, 21st June, 1940. The appointment of Honorary Captain J. C. Sangster is terminated.

To be Honorary Captains—Paul Franz Stratmann, 14th June, 1940; Frank James Alexander Juttner, 15th June, 1940; Richard Harper Formby and John Harvey Johnston, 22nd June, 1940; Ronald Lister Verco, 23rd June, 1940; Alec Douglas Cocks, 26th June, 1940; Frederic French Heddle, 6th July, 1940, and Basil Woodd Leach, 10th July, 1940. The appointment of Captain A. L. Tostevin is terminated.

Sixth Military District.

Australian Army Medical Corps.

Honorary Major J. B. Hamilton is appointed from the Reserve of Officers (A.A.M.C.) and to be Major (provisionally), 30th June, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Major—William Prout Holman, 6th June, 1940.—(Ex. Min. No. 169—Approved 27th August, 1940.)

WESTERN COMMAND.

Fifth Military District.

Australian Army Medical Corps.

To be Captain (provisionally)—Gerald Carew Moss, 30th June, 1940. Honorary Captain D. F. Mercer is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 30th June, 1940.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The following are granted commissions, on probation with the rank of Flight Lieutenant, with effect from the dates indicated: Sydney Morgan Lewis Dunstone, M.B., B.S., and Desmond Coleman Trainor, M.B., Ch.M., F.R.C.S. (Edin.), 22nd July, 1940; Godfrey Joseph Burke Baldwin, M.B., B.S., Stephen William Dobell-Brown, M.B., B.S., Wallace Hugh Fraser, M.B., B.S., D.P.M., Arthur Geoffrey McManis, M.B., B.S., Victor Robertson Meek, M.B., B.S., Rees James Riddell, M.B., B.S., D.T.M. & H., Raymond George Tonkin, M.B., B.S., and Frederick Ralph Wicks, M.B., B.S., 29th July, 1940.

Benjamin Epstein, M.B., B.S., is granted a commission on probation with the rank of Flight Lieutenant with effect from 5th August, 1940. Flight Lieutenant R. C. Gill, M.B., B.S., is transferred from the Reserve to the Active List with effect from 5th August, 1940. The notification appearing in the *Commonwealth of Australia Gazette* dated 18th July, 1940, concerning Roderick Lionel Jeffrey, M.B., B.S., is cancelled.

Reserve: Medical Branch.

Flight Lieutenant V. G. Renowden, M.B., B.S., relinquishes his commission with effect from 6th August, 1940.—(Ex. Min. No. 58—Approved 21st August, 1940.)

Correspondence.

MÉNIÈRE'S SYNDROME AND GOUT.

SIR: Dr. Dane reported a case in THE MEDICAL JOURNAL OF AUSTRALIA (August 3, 1940) and suggests that as his patient suffers from both Ménière's syndrome and gout, that the cause of the trouble is the gout. This is debatable.

The history tells that the patient, a man of fifty-six, ten years ago was seized with severe vertigo, and he was ill for several days. He had suffered from tinnitus in his right ear.

After the first attack of vertigo he was incapacitated for six months from vertigo. He had his tonsils removed; one was septic. Relief for two weeks, then the return of vertigo. Seen by another physician, it was diagnosed that the patient had had two attacks of gout; an anti-gout regimen kept the gout away for five years, also his vertigo.

Dr. Dane considers that gout was a major factor in the patient's illness; but he says he has not seen gout mentioned as a cause of vertigo or Ménière's syndrome. It may not be the cause, but Trousseau, Murchinson, Paget and Dyce Duckworth have described giddiness as a symptom of gout. John Hunter had syphilis and gout and suffered from attacks of intense giddiness, and on one occasion had to remain in the prone position for ten days. Trousseau quotes a case of vertigo from Boerhaave where an attack of gout entirely removed the tendency to vertigo for a long duration.

Dyce Duckworth says: "Among the symptoms depending on irregular gout is that of giddiness or vertigo." I emphasize the term irregular gout, as I claim to have shown that all examples of irregular gout are examples of allergy-gout in consequence of the patient having the gouty and allergic diatheses combined.

Dr. Dane says that the patient's father suffered from atypical gout, that is irregular gout, that is allergy-gout; the consequence is that the patient has probably inherited the gouty and allergic diatheses from his father.

Ménière's syndrome is due to allergy in many instances; and so in Dr. Dane's patient it was the allergy part of the allergy-gout that caused the endolymph or, as others suggest, the perilymph to be affected; not the gout part of the allergy-gout.

We know that in some cases of Ménière's syndrome the fluid of the labyrinth may be clear, while in some cases it may be mixed with blood. This is evidence that may be advanced to support the idea that the cause of Ménière's syndrome is allergy, for we know that the blood vessels in allergic subjects allow the serum to escape through their walls, while in some cases blood may come through the walls, just as it does in Henoch's purpura, which is due to allergy.

The removal of the tonsils did away with the bacterial allergens to which the patient was sensitive. The diet for his gout suited the gout and checked his allergy, so he kept well for five years; for if you overcome the allergy in allergy-gout the gout will cease, as it has done in my own case after forty years of allergy-gout.

This patient should be tested by an expert in allergy and should then be fed according to the results obtained, and should be desensitized by appropriate injections, and his endolymph trouble will probably cease.

His diet is most important, and from the experience I have gained from hundreds of cases in this town where I now reside, in a district that flows with milk and allergy, I should say that he should never eat pork or bananas, and he should beware of eggs and Australian wine. Finally, he should have every tooth X-rayed and all infected or loose teeth removed.

I have found that "Veramon" at bedtime is a help in these cases, as it is in epilepsy and migraine when due to allergy. But neither food nor drugs will take the place of desensitization.

Yours, etc.,

W. J. STEWART McKAY.

Lismore,
New South Wales,
August 21, 1940.

REORGANIZATION OF THE MEDICAL PROFESSION.

SIR: I was sorry indeed to miss Dr. Brown's essay on the reorganization of the medical profession. It was thoughtful and embodied one point of view.

May I recall to those interested that 2,000 years ago a Latin poet stated you may chase Nature away with a pitchfork, but she will always come home again. That observation is always in my mind, brought into thought by daily experience.

If they will read the essay "A Plea for a Positive Health Policy" by the late Sir Basil Blacket (a director of the Bank of England) they will find some agreement and much disagreement with Dr. Brown. Sir Basil Blacket shows that the national insurance system in Great Britain has not done what he hoped in the way of prevention of disease, but has, on the figures submitted, increased the quantity of disease and the cost thereof.

There are now fifty-seven bush nursing hospitals in Victoria, and more developing. Under Government regulation, I fancy, judging from action in other parts of Australia, there would have been none of this type. There would have been more large and expensive institutions to which everyone would have been forced or alternatively faced with the heavy expenses of a private hospital. And yet every one of these hospitals objects to any governmental control or assistance. They must follow the general rules of the Association. Otherwise they manage their own affairs, that is, they are decentralized. Yet each and every one has been created at the request of or with the support of local practitioners. They cost to build a fraction of the sum the large hospitals entail, and they offer superior accommodation and trained nurses, now some 150 in number.

If further evidence is wanted it can be found in the American Group Hospital Association. Commencing in 1933, this body had 3,000 subscribers. At the end of this year it is estimated that there will be 6,000,000. Yet it is organized and managed solely by the medical profession, who will receive money neither from the Government nor philanthropic agencies.

The Bush Nursing Association has received assistance from philanthropic organizations or it could not have attained its present dimensions. But it does not receive any governmental assistance for building and practically none for maintenance.

In the Royal Victorian Institute for the Blind the workers are paid the difference between what they earn and a living wage. It has fallen to my lot to prevent persons with good and even perfect sight obtaining admission in order to obtain the economic benefits.

I sympathize with Dr. Brown's desire for some organization, but from experience I am sure political management would be disastrous. Dr. Brown thinks we could get a commission like the Harbour Trust, which would keep clear of politics. But the Government, which holds the power of the purse, cannot be kept out of anything if they so desire. We have only too much evidence on that matter.

My sympathy is far more with Dr. Dale's suggestion that the profession should organize itself and keep the management in its own hands. I am sure that no government would agree to the limitation of the number of intending students. New Zealand has already so decided.

May I give an account of a frequent occurrence. A deputation travels 200 or 300 miles to Melbourne and interviews the President of the Victorian Bush Nursing Association, Lady Mitchell, and myself. They ask for a Bush Nursing Hospital for their district. The problem is investigated and they are told what the Bush Nursing Association can or cannot do to assist them. Had they waited on a Minister of the Crown, he might, and probably would, be just as or even more sympathetic, but would have informed them that he must consult the Cabinet. In perhaps two months' time they would get a sympathetic letter stating that the matter would be considered in next year's estimate. Then either nothing would happen or possibly an expensive hospital would be authorized. The present, and, I am afraid, any political system, is too cumbrous to undertake such work, and such changes must be made by the profession itself.

In conclusion I would like to refer to one possible incident in government control. When the antiseptic system developed, Bismarck was reported to be considering whether he would make failure to use the antiseptic system a criminal offence. Had all governments taken this step asepsis might never have been introduced. Medicine does not seem to develop properly under totalitarian political methods, and the wholesale dismissal of eminent men of science from their posts in Germany is a red letter warning to all. Freedom is essential if medicine is to develop as it should.

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,
Melbourne, C.1,
September 2, 1940.

Australasian Medical Publishing Company, Limited.

ANNUAL MEETING.

THE annual meeting of the Australasian Medical Publishing Company, Limited, was held at The Printing House, Seamer Street, Glebe, New South Wales, on September 2, 1940, Dr. T. W. Lipscomb, the Chairman, in the chair.

Directors' Report.

The report of the Directors of the company was as follows.

The Directors submit their report for the twelve months ended June 30, 1940, and the balance sheet as at June 30, 1940, together with the profit and loss account for the twelve months ended June 30, 1940.

During the year under review THE MEDICAL JOURNAL OF AUSTRALIA published articles which covered a wide range of subjects, and the standard of previous years was maintained. Paper rationing regulations will have the effect of reducing the number of pages during the coming year, but arrangements have been made to alter the style of the journal by the use of smaller type, which will obviate, to some extent, any reduction in the reading matter.

The output of the printing and publishing department compares favourably with that of previous years, but the paper rationing regulations will cause a reduction in the near future. This aspect is receiving careful attention. The work produced during the year was of the usual high quality and was mainly of an educational or scientific nature.

Depreciation amounting to £1,178 8s. 10d. was written off during the year, and an amount of £450 has been provided for taxation. The company's reserves are used in the business, and we consider that the state of the company's affairs is satisfactory.

Provision has been made for the payment of debenture interest for the twelve months ended June 30, 1940.

Dr. J. P. Major and Dr. Gregory Sprott retire from office by rotation in accordance with the Articles of Association (Article 39). They are eligible and present themselves for reelection.

T. W. LIPSCOMB,
Chairman.

July 29, 1940.

Election of Directors.

Dr. J. P. Major and Dr. Gregory Sprott were reelected to the Board of Directors.

Australian Medical Board Proceedings.

SOUTH AUSTRALIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1919 *to* 1935, of South Australia, as duly qualified medical practitioners:

Short, D. W., M.B., B.S., 1940 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Crosby, R. W. L., M.B., B.S., 1940 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Dutton, A. I., M.B., B.S., 1940 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Lloyd, H. A., M.B., B.S., 1940 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Lyons, H. E., M.B., B.S., 1940 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.
Levi, E. H., L.R.C.P. and S. (Edinburgh), L.R.F.P.S. (Glasgow), 1939, Adelaide.

The following additional qualifications have been registered:

West, Robert Frank, Nailsworth (M.B., B.S., Adelaide, 1935), M.R.C.P., London, 1937, M.R.A.C.P., 1939.
McLarty, Thomas Leslie, Adelaide (M.B., B.S., Adelaide, 1935), D.O.M.S., London, 1937.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Berry, Annie Hayes, M.B., Ch.B., 1937 (Univ. New Zealand), The Women's Hospital, Crown Street, Sydney.
Coolican, Michael Raphael, M.B., Ch.B., 1922 (Univ. Dublin), c.o. British Medical Agency, Macquarie Street, Sydney.
Elder, Russell Exon Butchart, M.B., B.S., 1920 (Univ. Melbourne), F.R.C.S. (Edinburgh), 1925, 63, Elizabeth Bay Road, Elizabeth Bay.
Hudson, Rodney James, M.B., 1940 (Univ. Sydney), Sydney Hospital, Sydney.
Little, Murray Frederick, M.B., B.S., 1939 (Univ. Sydney), District Hospital, Manly.
Silverman, Herbert Nathan, M.B., B.S., 1937 (Univ. Melbourne), The Women's Hospital, Crown Street, Sydney.

The following additional qualifications have been registered:

Millard, Philip Thornton, Orange (M.B., 1933, B.S., 1937, Univ. Sydney), F.R.C.S. Edinburgh, 1938.
Watt, John Langston, Tamworth (M.B., 1933, M.S., 1938, Univ. Sydney), F.R.A.C.S., 1940.
Windeyer, John Cadell, Sydney (M.D., Ch.M., Univ. Sydney, L.R.C.P. London, M.R.C.S. England), F.R.A.C.S., 1927, F.R.C.O.G., 1932.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Brett, Alan Sidney, M.B., B.S., 1940 (Univ. Sydney), 30, Tranmere Street, Drummoyne.

Medical Appointments.

The following honorary appointments have been made at Saint Vincent's Hospital, Sydney. Physicians: Dr. O. A. Diethelm, Dr. J. P. Tansey, Dr. R. J. Taylor, Dr. R. Jeremy; Assistant Physicians; Dr. M. J. Slattery, Dr. J. Sherwood, Dr. Bruce Hall, Dr. M. J. McNamara, Dr. R. L. Spedding, Dr. P. J. Markell; Surgeons: Dr. V. M. Coppleson, Dr. W. T. D. Maxwell, Dr. I. D. Miller, Dr. V. J. Kinsella; Assistant Surgeons: Dr. E. G. MacMahon, Dr. L. May, Dr. W. S. McGrath, Dr. F. H. Mills; Gynæcologists: Dame C. E. D'Arcy, Dr. F. Brown Craig; Assistant Gynæcologists: Dr. D. S. Foy, Dr. M. B. Fraser; Urological Surgeon: Dr. W. Perry; Ear, Nose and Throat Surgeons: Dr. H. S. Marsh, Dr. J. J. Woodburn; Assistant Ear, Nose and Throat Surgeons; Dr. W. R. Beavis, Dr. H. B. Harwood, Dr. H. S. Kirkland, Dr. B. Blomfield; Ophthalmic Surgeons: Dr. F. G. A. Pockley, Dr. H. Odillo Maher, Dr. A. L. North; Assitant Ophthalmic Surgeons: Dr. A. Odillo Maher, Dr. A. E. F. Chaffer, Dr. J. A. Pockley, Dr. T. M. Armstrong; Orthopædic Surgeon: Dr. D. J. Glissan; Assistant Orthopædic Surgeon: Dr. W. H. Smith; Pathologist: Dr. A. H. Tebbutt; Dermatologist: Dr. G. B. Lindeman; Assistant Dermatologists: Dr. W. E. Ryan, Dr. E. M. Will, Dr. G. R. Gillies, Dr. W. K. Myers; Radiologists: Dr. B. P. Anderson Stuart, Dr. A. F. Oxenham, Dr. D. G. Maitland; Psychiatrist: Dr. W. R. Page; Radiotherapist: Dr. C. de Monchaux; Instructor in Practical Anæsthesia: Dr. H. J. Daly; Assistant Instructor in Practical Anæsthesia: Dr. S. V. Marshall; Anæsthetists: Dr. J. F. McColloch, Dr. W. G. Holt, Dr. L. MacMahon, Dr. L. T. Shea, Dr. C. N. Paton; Clinical Assistant to Medical Out-Patient Department: Dr. J. Christie; Clinical Assistant to Surgical Out-Patient Department: Dr. W. G. Holt; Clinical Assistant to Dermatological Department: Dr. Edna L. Nelson; Clinical Assistant to Radiotherapy Department: Dr. Nell Farrar.

Books Received.

"Recent Advances in Sex and Reproductive Physiology", by J. M. Robson, M.D., D.Sc., F.R.S.E., with an introduction by F. A. E. Crew, M.D., D.Sc., F.R.S.; Second Edition; 1940. London: J. and A. Churchill Limited. Demy 8vo, pp. 342, with 62 illustrations. Price: 15s. net.

"Modern Dietary Treatment", by M. Abrahams, M.A., M.Sc., and E. M. Widdowson, B.Sc., Ph.D.; Second Edition; 1940. London: Baillière, Tindall and Cox. Crown 8vo, pp. 413. Price: 10s. 6d. net.

Medical Research Council of the Privy Council. Special Report Series, No. 238: "The Structure of Teeth as Shown by X-Ray Examination", by J. Thewlis; 1940. London: His Majesty's Stationery Office. Medium 8vo, pp. 82, with illustrations. Price: 2s. 6d. net.

Diary for the Month.

SEPT. 17.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 18.—Western Australian Branch, B.M.A.: Branch.
SEPT. 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 25.—Victorian Branch, B.M.A.: Council.
SEPT. 26.—South Australian Branch, B.M.A.: Branch.
SEPT. 26.—New South Wales Branch, B.M.A.: Branch.
SEPT. 27.—Tasmanian Branch, B.M.A.: Council.
SEPT. 27.—Queensland Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associate Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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